The

DENTAL JOURNAL

of

AUSTRALIA

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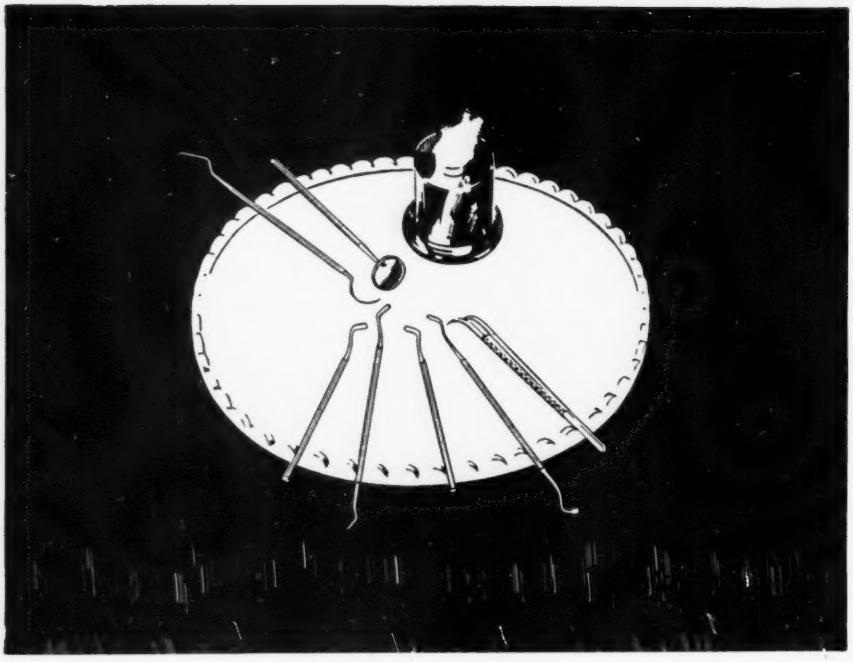

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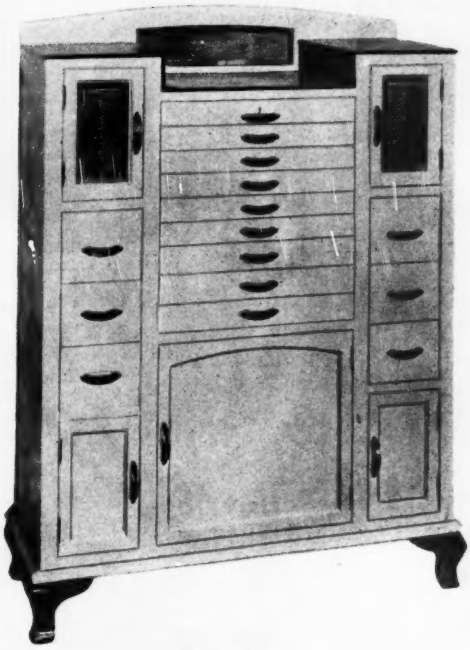

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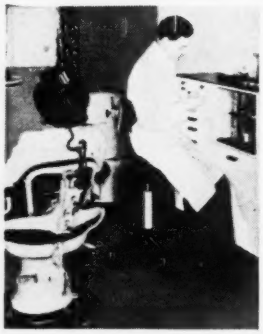

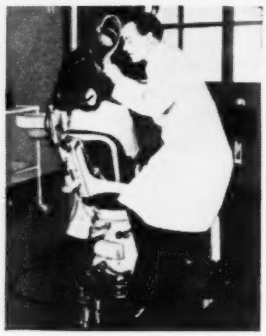

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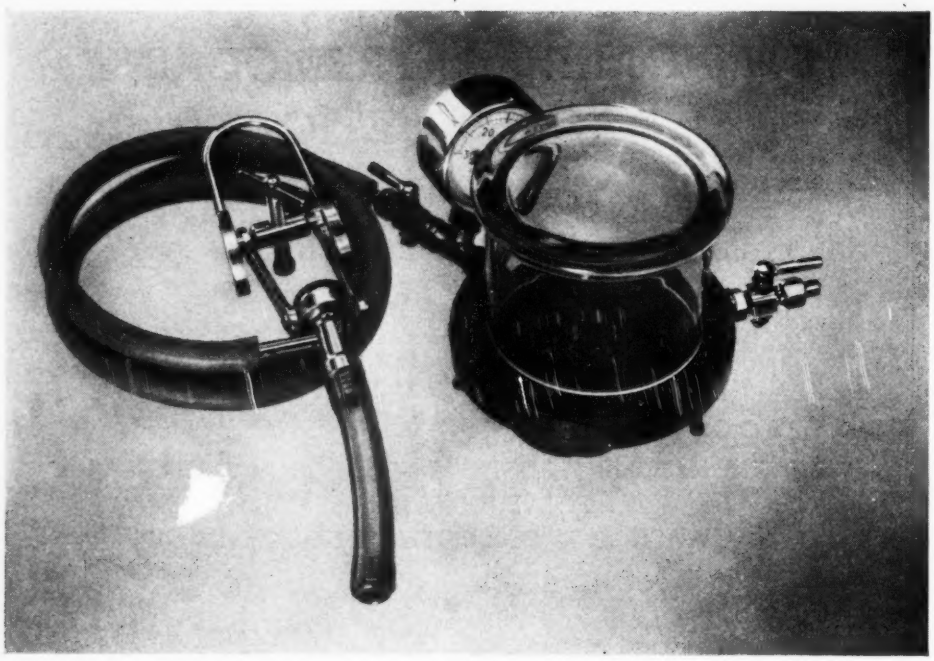

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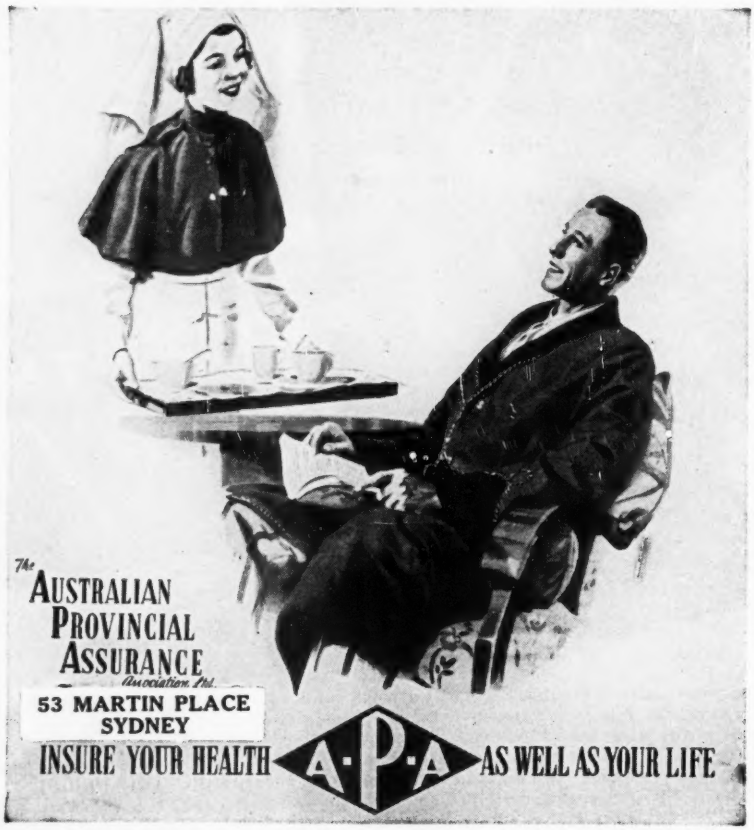

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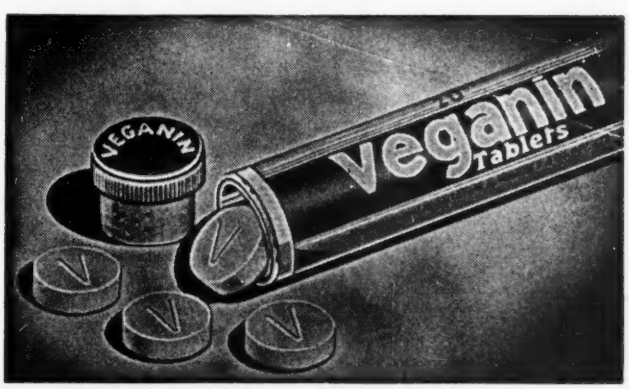

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G. & G. Medium
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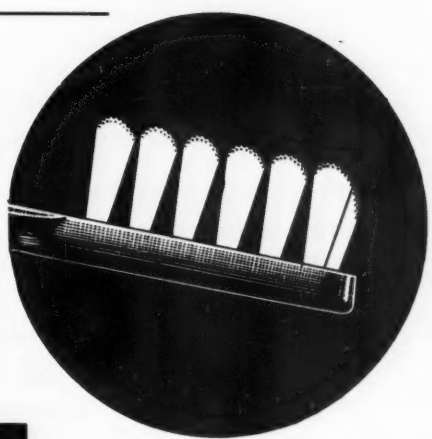

NADA

THE DENTISTS'-DESIGN TOOTHBRUSH

AT CHEMISTS EVERYWHERE

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G. & G. Quick Setting
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Let these typical results convince you

Test	G. & G. Medium Alloy	Australian Standard T2
Mixing time in seconds	50	max. 90
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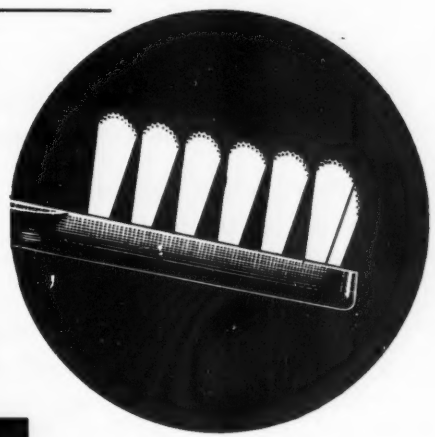

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The Formation of Bone*

M. Jolly, M.D.S. (Syd.).†

QUELLES SONT LES CAUSES DE L'EVOLUTION OSTEOBLASTIQUES DES FIBROBLASTES? PERSONNE NE NOUS LE DIT.

(Leriche and Policard, 1934.)

1. Growth Initiation.

(a) *The evocation of bone formation.*

Attempts to analyse the factors responsible for the development or repair of bone have been numerous and there has been active discussion on the possibility of the existence of a specific osteogenic substance. Most investigations of this subject have involved the study of the survival or formation of bone tissue from bone-grafting materials. From basic reasoning it would seem to the writer that at the earliest stages of embryonic development there must exist certain specific agents which cause the differentiation of primitive cells into those of the primary tissues, and there is the possibility that these agents are retained throughout life. They may be in the form of chemical substances and thus could have either an intracellular or extracellular existence, suggesting the possibility of extracting and isolating such compounds. Again, the differential growth potential could be a function of genetic patterns and therefore entirely intracellular in location and effect.

Regarding bone regeneration, there are at present two theories. Proponents of the one hold that bone formation is dependent upon local physical and chemical changes including hydrogen ion concentration and possibly the presence of a specific osteogenetic substance. Those who subscribe to the other believe that

> the osteoblasts derived from the primitive connective tissue, the endosteum, the periosteum, or the Haversian canals may survive transplantation and are capable of reproducing bone.[1]

In 1938 Levander[2] published the results of a series of investigations on bone regeneration. In this paper he evaluated the results of earlier investigations of the osteogenetic power of the periosteum. A dominating role in bone regeneration had long been ascribed to this tissue as a result of experiments showing bone formation after transplantation of isolated periosteum into soft tissues. Levander noticed a lack of uniformity in the results of these experiments for, while some authors had found bone to be formed with fairly great regularity, others had reported large series of experiments in which no single case of bone formation could be demonstrated. He showed a definite relationship between the results obtained and the type of tissue transplanted. His explanation was based on the fact that the periosteum is made up during skeletal growth of two well differentiated layers, an inner one (the cambium layer) and an outer layer (the adventitia) which consists of ordinary connective tissue poor in cells.

> At the termination of skeletal growth the cambium layer disappears, and the cell deficient adventitia alone remains.[2]

From experimental investigations he showed that

> on periosteal transplantation bone formation takes place only in animals the skeletons of which are still in the process of growth.[2]

He concluded therefore that it was impossible to attribute any specific importance to the bone forming capacity of periosteum. Levander transplanted pieces of fully differentiated, hard, living bone tissue free of periosteum taken from the middle of the radius or ulna of rabbits either subcutaneously or intramuscularly. Fully grown as well as young animals were used and in all of them autoplastic grafts were transplanted. He noted the early proliferation of cells of mesenchymal appearance—so called vascular mesenchyme— around the graft, followed in all cases by the early formation of new bone tissue, and occasionally a cartilaginous islet was seen. He observed the early death of the bone cells of the graft so that at the end of about two weeks all bone graft tissue was dead. Nowhere were there signs of cell division within the graft. It was noted that new bony tissue was formed in the mesenchymal tissue and even at some distance from the graft.

In the hope of discovering the presence of a substance with bone-forming properties, Levander made aqueous extracts of bone tissue and injected them into the muscles of rabbits, but all the results were negative. He then made intra-muscular injections of alcohol autoextracts of bone and callus tissue and in 22% of the group of seventy animals observed the formation of cartilage or bone. In a controlled series of 80 tests sixty animals were injected with alcohol solutions of the same concentration and twenty were given injections of extracts of connective tissue, striated and smooth muscle. In these cases an abundance of newly formed connective tissue was observed but never cartilage or bone. As a result of these experiments the author concluded that

> bone regeneration takes place as the result of some specific bone forming substance activating the non-specific mesenchymal tissues.[2]

*Portion of thesis submitted in part fulfilment for the degree of Master of Dental Surgery, University of Sydney.

†Lecturer in Operative Dentistry, University of Sydney.

Levander[3] in 1940 made subcutaneous autoplastic bone marrow grafts into both young and adult rabbits and obtained bone formation, and again concluded that the bone was not produced from the marrow itself but from the mesenchymal tissue around it which had been induced to form bone by some osteogenic substance in the marrow. Claims for an isolation of an "alcohol-soluble osteogenetic substance" derived from bone marrow were made by Levander and Willstaedt[4] in 1946.

In 1941 it was demonstrated by Bloom, Bloom and McLean[5] that the marrow reticulum cells of female pigeons differentiate into osteoblasts during the formation of medullary bone and become osteoclasts during bone resorption. This behaviour suggested that bone could be produced from the reticulum cells of the marrow in the absence of both endosteum and formed bone, if the proper environment were present.

In an introduction to his report of the existence of an "organizer" of the growth of bone, Lacroix[6] gave an excellent description of the mode of growth of long bones of rabbits, in the region of the metaphysis. He described the important function of the "perichondrial bony ring of the ossification groove" in forming and shaping the bone in this region during elongation of the shaft. The reason for describing this structure was that if true "induction" of bone tissue by extracts or grafts from the region of the epiphyseal cartilage were to take place then some indication of this ring should also be found. In the first set of experiments the central part of a growth cartilage was used as an auto-implant in the brain of the experimental animals (young rabbits). Lacroix reported the continued growth of the cartilage and the accumulation around it of cells "which seem to be ordinary fibroblasts". Some of these cells had taken part in the formation of endochondral trabeculae of bone which in places were being absorbed and replaced by haemopoietic marrow. A thin bony lamella was seen which had all the characteristics of the "perichondrial bony ring of the ossification groove".

In a second experiment

> A rod of hyaline cartilage was taken from the rib of a rabbit and put into the upper extremity of the tibia of another young rabbit in such a manner that it pierced the centre of its proximal growth cartilage.[6]

The growth cartilage went on proliferating and after a few weeks "the grafted cartilage was the site of a typical endochondral ossification".

> The serial sections proved that the phenomenon did not follow an invasion of the graft by the host cartilage cells, but that it was the consequence of the contact of both tissues. The growth cartilage behaved as an inductor and imposed its organization upon a tissue which, had it been grafted alone anywhere else, would have remained a piece of hyaline cartilage.[6]

In a further experiment Lacroix attempted to prove the existence in the growth cartilage of an organizer. Alcoholic extracts from the growth cartilage were injected into the thigh muscles of another rabbit. "Forty-one days afterward, a large osteoma was found in the thigh." On examination it showed growth cartilage with its characteristic arrangement and partially surrounded by the perichondrial ring of the ossification groove.

Pfeiffer[7] reported the development of bone from transplanted marrow in mice in 1948. He removed marrow from the femurs of one group of mice and implanted it in the anterior chambers of the eyes, testes and ears of other mice. In a large percentage of cases bone developed in the marrow that had been transplanted to the anterior chamber of the eye and between the seminiferous tubules of the testes (regions of low resistance to grafting) but in no case did bone form in the subcutaneous areas of the ear (region of high resistance to grafting). The bone was surrounded by a typical endosteum with numerous osteoblasts and occasional osteoclasts. Immediately outside of this was a layer of loose connective tissue containing numerous plasma cells. The plasma cells were seen to transform into fibroblasts and the author suggested that the plasma cell may be an intermediary in the formation of osteoblasts from the reticulum cells. In these experiments the myelogenous and haemopoietic cells did not survive. Because of this and "since the amount of marrow transplanted was too small to contain effective quantities of the osteogenic material[7]" the author concluded that the marrow reticulum cells were the prime factor in osteogenesis either by differentiation or the production of an osteogenic material.

Some pertinent experiments were reported in 1947 by Hancox[8], who criticized the theory of the existence of a specific bone organizer. He transplanted bone grafts from embryo chick to the chorioallantoic membrane of another embryo (10 days incubation) and followed a technique whereby the transplanted fragments could be directly examined microscopically in the living condition at any time. One of the most striking observations was the rapidity with which "a complete circulation became established within the live fragments".[8] Thirty-two of forty successfully transplanted live fragments were extensively vascularised within 18 hours, and the remainder within 48 hours, this being verified by the observation of definite movement of blood corpuscles along the vessels of the graft. There were two possibilities regarding the development of the new circulation within the graft. Firstly it could have arisen from capillary sprouts which pushed their way through the Haversian systems or, secondly, new vascular buds may have grown

up to and fused with the cut ends of the graft vessels. Hancox favoured the second explanation and stated that the time factor was in its favour.

The bone fragments were studied for as long as 10 days and it was stated that "both resorption and deposition of matrix occurred".[8] Boiled fragments were also transplanted and in no case was vascularization observed within 48 hours but vessels did finally appear within the bone. No bone formation occurred within the 10 days observation period.

Hancox concluded

> thus, under the present experimental conditions, which, essentially predicate a highly reactive and vascular graft bed, it is safe to assume that transplanted fragments of live bone not only survived, but that, through the agency of their surviving cells they deposited fresh bone substance. The necessity for postulating a specific osteogenic inductor does not, therefore, arise in these experiments.[8]

In attempting to evaluate the various theories advanced on the subject of evocation of bone formation it would appear that insufficient experimental and clinical evidence is at present available to reach a definite conclusion. However certain authors[2-5] and particularly Lacroix[6] have put forward very convincing evidence for the existence of a specific bone organizer. The only criticism that can be made of Lacroix is that very few animals were used in his experiments and no mention was made of controls. Criticism of this theory by Hancox[8] was somewhat ineffective for although he proved the survival of cells of the graft in an ideal environment this did not necessarily preclude the action of some osteogenetic substance.

(b) *Physiology.*

Introduction. The several types of connective tissue in the body all consist of two chief components: (1) living cells and (2) non-living organic intercellular substances which are produced by the cells and lie between and outside them. The connective tissue which normally becomes calcified contains the same basic components as an uncalcified tissue, and the only fundamental difference is that its organic intercellular substance becomes impregnated with mineral salts. Ham[9] has drawn attention to the fact that, although the intercellular substance of connective tissue is non-living, its calcification makes "the nutrition, growth, repair and grafting of the tissue both more difficult and more complicated than is the case in an uncalcified tissue".[9] Far from being almost completely stable, bone is one of the most labile tissues of the body, and one in which every change of tissue metabolism, diet, and function is recorded. Bone not only reflects changes in the rest of the body, but also plays its part in bringing them about. Thus for a complete understanding of the behaviour of bone in relation to the rest of the body it would be necessary to know exactly the function of the bone cells and the precise physical and chemical state of the inorganic constituents in relation to their organic framework and local environment.

The bone cell. It is universally accepted today that the bone cell is responsible for the formation of bone and its continued existence. Leriche and Policard[10] challenged this view and regarded the osteogenic cells as resisting an advancing osseous mass of extracellular origin, leaving captives behind them to be enveloped and form the osteocytes of mature bone. However, it was generally accepted when Newton[11] stated in 1939 that

> there can be no doubt that the capacity for forming bone is inherent in the bone cell itself at a very early stage of development.

Nourishment of the bone cells. Food and oxygen are brought to the body tissues by arteries and released to the cells through the thin walls of the arterial capillary network. Similarly waste products pass into the venous capillaries and so to the larger veins. It has been pointed out by Ham[9] that the great majority of body cells are not in direct apposition with capillaries but are separated from them by intercellular substance and other cells and therefore there must be some efficient mechanism "beyond the capillaries" for the conveyance of nutrients and oxygen from capillaries to the cells and waste products from cells to capillaries. In order to evaluate this mechanism he has compared the mode of nourishment of calcified tissues with that of the non-calcified tissues.

Mechanisms in dermis. From observation of a capillary loop in the dermis just below the epidermis it may be seen that the capillary lies in the intercellular substance of connective tissue. Connective-tissue cells are scattered throughout the intercellular substance. Ham described three ways by which the cells of the non-vascular epidermis above may be nourished.

(1) *By circulation of tissue fluid.* Until the present time the greatest importance has been attached to this form of nourishment of the cells and it has been assumed that tissue fluid, as a dialysate of plasma, is more or less continously produced by the arterial blood capillaries and just as continuously absorbed by venous capillaries and by lymphatic capillaries. Accordingly there must be an actual circulation of the tissue fluid and this requires among other factors that the intercellular substances be freely permeable to tissue fluid or that they present a net work of clefts or passageways for the conveyance of the tissue fluid.

(2) *By diffusion through tissue fluid.* It has been doubted by several authorities whether a very substantial circulation of tissue fluid can occur under normal conditions. It could, however, if it bathes the intercellular substance and the cells, act as a medium for the diffusion of food and waste substances.

(3) *By diffusion through gels.* For many years the intercellular substance of connective tissue has been said to comprise two elements, (a) the protein fibrous elements (collagenic, reticular and elastic fibres), embedded in (b) the so-called ground and cement substances. Recently Meyer[12] has reported that these latter substances are more of the nature of carbohydrates than proteins and has termed them mucopolysaccharides. These substances may be divided broadly into two groups, the sulphated types and non-sulphated types. Hyaluronic acid is the common example of a non-sulphated kind and chondroitin sulphuric acid the main sulphated kind. Hyaluronic acid apparently can exist as a sol, as in aqueous humour or synovial fluid, or as a gel, as in the ground substance of areolar tissue. The gels formed by the sulphated types are, in general, firmer than those formed by hyaluronic acid; hence the sulphated types are often called "cement substances".

Ham has envisaged the cement substances as having a dual function; they serve a supporting role but in addition have the important function of providing a medium through which diffusion can readily occur between capillaries and cells.

> Indeed it seems very probable that this is a very important function of the mucopolysaccharides, and it is perhaps significant with regard to the impaired nutrition of the tissues of the aged that the concentration of mucopolysaccharides is not so great in many of their connective tissues as it is in those of the young.[9]

In the dermis as well as in some other uncalcified connective tissues it is conceivable that all three mechanisms operate in the nourishment of the cells.

The mechanism in cartilage. Like other connective tissues cartilage consists of cells and non-living organic intercellular substance. The cells live in lacunae surrounded by intercellular substance which consists of [illegible]en fibres embedded in the mucopolysa[illegible]aride cement substance, chondroitin sulphuric acid. Typical hyaline cartilage is avascular like the epidermis of skin and similarly its cells must be nourished by one of the mechanisms that operate beyond the capillaries. The one that operates in cartilage is that of diffusion through a gel.

Although water is present in the intercellular substance and is indeed the dispersion medium of the gel, it also forms an integral part of the gel structure and must be thought of as being different to free tissue fluid. The gel leaves no room in the substance of the intercellular material for free tissue fluid. Thus, although the outside of a given cartilage may be bathed in free tissue fluid the individual cells cannot be and rely on the diffusion of food and waste products through the gelled intercellular substance for the maintenance of their vitality. The understanding of this fact is necessary for the interpretation of events following the calcification of cartilage.

The precise details of the calcification of cartilage are not fully known. Maximow and Bloom[13] merely described calcification as one of the "normal regressive changes of cartilage" and state that this process usually precedes the replacement of cartilage by bone. Weinmann and Sicher[14] state that the calcification of cartilage is preceded by degeneration of the chondrocytes. This causes a drop in the carbon dioxide concentration parallel to a drop in cellular metabolism and thus a rise in the pH level. Such a hypothesis proposes a passive role of the chondrocytes in the calcification of cartilage. The chemical and histochemical investigations by Sylven[15] into the physiological role of chondroitin sulphuric acid in cartilage would suggest a constant depletion of this substance with aging. This fact must be correlated with a number of different factors. From infancy to old age a continuous decrease in the number of cells is found in articular cartilage and at the same time the content of calcium increases. Sylven[15] states that there is also a continuous process of cell degeneration accompanied by many changes such as calcification, pigmentation and fibrous metaphasia of cartilage. On the nutrition of cartilage Sylven stated that

> It is generally agreed that cartilage, being largely devoid of vessels, gets the nutritive fluid by diffusion from the blood vessels of the perichondrium. This means that the fluid must pass through the interstitial substance to reach the chondrocytes.[15]

With articular cartilage the diffusion is probably through the synovial fluid.

As early as 1935 Logan[16] found that calcification of cartilage was preceded by a loss of organic sulphates, and as Sylven[15] has been unable to detect any other sulphates in cartilage it may be assumed that Logan was in fact estimating chondroitin sulphuric acid content.

In a later report of cartilage analysis Sylven[17] related chondroitin sulphuric acid content to alkaline phosphatase. His conclusions were:

> (1) During the early embryogenesis of cartilage, the gradual increase in content of chondroitin sulphate is accompanied by a corresponding decrease in alkaline phosphatase.
>
> (2) Normal adult hyaline cartilage, which does not ossify, contains in its intercellular matrix a large amount of chondroitin sulphate, but little or no alkaline phosphatase.

(3) The normal embryonal ossification process is characterized by a rapid local disappearance of chondroitin sulphate, and concurrently by the appearance of large amounts of alkaline phosphatase.[17]

It would appear from these findings that removal of the acid chondroitin sulphate is a prerequisite for the action of the bone-forming osteoblasts. There has been the suggestion that the presence of chondroitin sulphate is the main protection against calcification in cartilage but Sylven[17] reported physiological decrease in the content of chondroitin sulphate in fibrous metaplasia of cartilage. Such condition is not necessarily combined with calcification.

Sufficient evidence is available to show that the healthy state of hyaline cartilage must in some way be associated with a high content of chondroitin sulphuric acid in its intercellular matrix. Also, the destruction and removal of this substance seems to be a necessary link in the chain of events producing an alkaline medium, which is a prerequisite for the action of alkaline phosphatase. Ham[9] postulated a theory for calcification of cartilage in which the chondrocytes were allotted an active role. He stated that when cartilage cells mature and become very large they secrete alkaline phosphatase which acts to increase the concentration of phosphate ions about them. The fluid of the gelled intercellular substance is probably already almost saturated with calcium salts and the increase in phosphate ion concentration is sufficient to cause precipitation. It is thought that the intercellular substance has an affinity, either chemical or physical, for the precipitate, so, as it forms, it settles into the gel structure. Ham has claimed that "This almost completely destroys the gel's ability to serve as a means for diffusion." Consquently when the intercellular substance of cartilage becomes calcified the pathway for the conveyance of food to the cells and waste products back to the blood is removed. The result is death to the cells.

In evaluating the theories of Sylven[15, 17] and Ham[9] it is clear that they both regard calcification of cartilage as being closely associated with death of the chondrocytes. What is still obscure is whether the chondrocyte itself produces alkaline phosphatase which in turn causes depolymerisation of chondroitin sulphuric acid or whether the chondroitin sulphate remains to be inactivated by precipitation of mineral salts in the gel structure. Part of the answer to this problem may have been supplied by Howard[18] in the following: Uncalcified cartilage is metachromatic while calcified cartilage is not. This was previously interpreted by Sylven[15, 17, 19], who used the metachromatic staining reaction for the histochemical assay of chondroitin sulphate, to mean that the chondroitin sulphate of the cartilage matrix was removed during the conversion to the calcified state. However, when calcified cartilage is treated with 1 per cent. nitric acid for five minutes it becomes highly metachromatic.[18] Thus the chondroitin sulphate of the matrix had not disappeared when the lime salt deposition occurred; it had merely been inactivated. Similar reactions were noted with osteoid and calcified bone and Howard[18] concluded that there is also a polysaccharide in calcified bone. Most authors assume that chondroitin sulphuric acid is produced by the cartilage cells but the present writer has been unable to find any direct evidence for this.

The mechanism in bone.

The calcification of the intercellular substance of connective tissues may be regarded as an attempt by nature to provide a strong rigid tissue. As Ham has pointed out, nature was not very successful in the case of cartilage for although the strength and rigidity are temporarily increased, the cartilage cells die, and the calcified intercellular substance becomes eroded and ultimately disappears. It is not entirely understood why the persistence of fully formed calcified intercellular substance is dependent upon the vitality of its cell content. Ham suggested

It seems probable that without the continued production of phosphatase by the cells the mineral deposit tends to go back into solution and, when this occurs in the living body, the organic intercellular substance in which it was deposited simultaneously dissolves away.[9]

The problem of producing a rigid, weight-bearing tissue in which the cells could still be nourished and maintained was solved with the evolution of bone as seen in the higher animals and especially in the human.

Although very similar to hyaline cartilage bone differs in one important respect. Instead of possessing smooth cell outlines like chondroblasts the osteoblasts, which give rise to the intercellular substance of bone, have many long and branching cytoplasmic processes which extend out from the cell body. These processes communicate not only with others of the same cell, but also with the processes of neighbouring cells thus forming a continuous network in a given area. Hence when the osteoblasts lay down intercellular substance the finished product not only encloses the cell bodies but also their branching and anastomosing processes. The processes can then be withdrawn towards the cell bodies leaving a network of tiny canaliculi which communicate on the one hand with lacunae and on the other with surfaces where tissue fluid is available. Thus the tissue fluid may come to occupy the network of canaliculi and all the space in the lacunae not taken up by the cell bodies of the bone cells. Because

of the very fine calibre of the canaliculi and the rigid nature of the surrounding bone it seems unlikely that any substantial degree of circulation could take place in them. More probably this is an example of the second basic mechanism for nutrition beyond the capillaries, i.e. diffusion through tissue fluid. The amount of tissue fluid available as a medium for diffusion is necessarily very small because of the fine calibre of the canaliculi; accordingly the mechanism cannot operate over any but very short distances. The following measurements between lacunae and capillaries have been given

> Both in compact and cancellous bone, the great majority of bone cells are no more than one-tenth of a millimeter away from a capillary and only an occasional one exists one-quarter of a millimeter away from a capillary.[8]

Blood supply to the fine structure of bone.

(1) *Cancellous bone*: The capillaries of the marrow spaces provide tissue fluid to bathe the trabeculae of cancellous bone. Within a trabecula the canaliculi of each lacuna communicate with those of adjacent lacunae and those near the surfact extend to the exterior of the trabecula and so permit tissue fluid to enter the anastomosing system of canaliculi. If trabeculae are more than one-fifth of a millimeter in thickness they generally have blood vessels disposed in canals near their mid-line. Starr[20] has pointed out that the larger trabeculae contain blood vessels and that these blood vessels are surrounded by concentric lamellae. Thus it may be seen that nutrient substances to reach the lacunae in the middle of a trabecula must diffuse along the canalicular system for approximately one-tenth of a millimeter in order to provide the more deeply disposed bone cells with nourishment.

(2) *Compact bone*: The compact bone is made up of Haversian systems as described in a section of this review not published in this Journal, and thus the bone cells lying between concentric lamellae are no further away from capillaries than are those of cancellous bone. The diameter of each Haversian system is necessarily limited to a maximum of approximately one-fifth of a millimeter so that the mechanism for providing nourishment does not have to operate over distances greater than one-tenth of a millimeter to supply the cells of the outermost layers. It can be appreciated from these measurements that the capillary bed of compact bone is of an extraordinary magnitude.

Ham[9] pointed out that there are two important reasons why the growth, repair and grafting of bone are relatively complicated:

(1) The canalicular mechanism which keeps the cells of a calcified connective tissue alive is at best an inefficient mechanism, and its effective operation requires that no bone cell is ever more than a fraction of a millimeter from a capillary.

(2) Because of the secretion of phosphatase by the osteoblasts, the organic intercellular substance calcifies almost immediately after its formation. "This feature of bone also prevents it from growing in as uncomplicated a way as uncalcified tissues".[9]

2. Growth of Bone.

(a) *General principles of bone growth.*

Uncalcified tissues such as the skin can increase in size by interstitial growth. These tissues can literally expand at any or all points within their substance with cells here and there dividing and forming new intercellular substance. Whether or not interstitial growth can take place in a tissue is dependent upon the nature of its intercellular sustance. This type of growth is only possible if the intercellular substance is sufficiently plastic to permit its being steadily expanded and reshaped from within. Hyaline cartilage exhibits interstitial growth and it is by virtue of this that most bones increase in length.

Because the intercellular substance of bone becomes calcified almost as soon as it is formed, it is not sufficiently plastic to permit interstitial growth. Bone can only increase in size by means of new bone being deposited on one or more of its surfaces by osteoblasts. This is called *appositional growth* and is the *only way that bone can grow*. One of the most important advances in bone biology is the conclusive demonstration that there is no interstitial bone growth. Since all growth of bone is a surface phenomenon there must be cells present on bone surfaces with an osteogenic potential.

Histologically there are two phases in the formation of bone: primary ossification and secondary ossification. These phases overlap and may only be distinguished from one another by the texture of the osseous tissue formed. Therefore there are two types of bone structure:

(i) Primary, immature or coarse-fibred bone.

(ii) Secondary, mature, fine-fibred or lamellar bone.

(i) *Primary bone.*

> In the lower animals, the human foetus and new-born skeletons, the collagen fibres of mammalian bone are coarse and very irregularly disposed. The associated cells are large, numerous and irregular. Such bone is termed primary or coarse-fibred bone.[20]

Phylogenetically and ontogenetically the bone formed in the early stages of bone development differs in its structure from that of mature bone. This difference is manifest in both the cellular and intercellular elements. The bundles of fibrils in the matrix of immature bone are coarse (averaging 12 to 15 microns in diameter) and decussating in all directions and the osteocytes are numerous but irregular in shape and arrangement and have but few anastomosing processes. These cellular processes are short and stout and perforce the canaliculi are wider and shorter than in adult bone. This approximation of the bone cells is often so extreme that large irregular lacunae may be seen containing numerous bone cells separated only by fibre-bundles. The total mineral content of the ground substance of primary bone is lower than that of secondary bone and the clusters of calcium phosphate crystals are larger and less regularly distributed.[14] Weinmann and Sicher[14] state that immature bone is always arranged in trabeculae and is, therefore, always spongy bone, depending upon the marrow for its nourishment. However Starr[20] claims that the trabeculae shows numerous wide longitudinal canals of the Haversian type similar to those in the adult, except that they are elongated in section and not circular, with wide infrequent intercanalicular anastomoses. The short trabeculae of this tissue bound numerous polyhedral marrow spaces with centrally located blood vessels and containing many large active osteogenic cells. It may therefore be concluded that primary bone is a very active biological tissue. It must be emphasised that although this is a primitive tissue mitotic division of the osteocytes does not occur and it has been demonstrated conclusively that there is no interstitial bone growth. The trabeculae grow by the addition of new bone laid upon the old, i.e. by appositional growth. In the developing skeleton, bone forms everywhere primarily as spongy bone which precedes secondary bone. By the fourth year of life primary bone has been almost entirely replaced by secondary adult bone but in some situations, e.g. at tendon attachments, primary bone persists throughout life.

In repair of fractures in the adult, primary bone is first formed and later replaced by secondary bone. Also in certain pathological conditions such as ossifying fibrous tumours and osteogenic sarcoma the newly formed bone is usually of the immature, coarse-fibred type. It may be said that, except in the case of physiologic reconstruction of secondary bone in conjunction with concomitant resorption, new formation of bone is always of the primary type at first and this is later replaced by secondary lamellar bone.

(ii) *Secondary bone* is the normal type seen practically throughout the skeleton after the fourth year. The whole structure has a more ordered and uniform appearance, the canals are narrower, the fibre-bundles are thinner and much more regularly disposed, and the enclosed cells have diminished in size.

Intermediate types of bone.

Bone, intermediate in structure between primary and secondary bone, may often be observed.[20] During bone deposition a type of bone may be formed having fine fibres but irregular structure later to be resorbed and replaced by secondary bone. Also the process of resorption which is well illustrated at the broken bone end during repair of a fracture results in a macroscopic rarefraction in which the bone shows irregular trabeculae and a large soft-tissue content somewhat similar to primary bone. However, microscopic examination will show the remnants of the true lamellar structure. Similarly, an intermediate stage may be observed during the reconstruction of callus after a fracture. In some pathological conditions intermediate types of bone are formed.

The development of bone begins early in the life of the foetus and growth and reconstruction continue until terminated by the death of the individual.[20]

There is merely one type of development of bone tissue; the terms endochondral and intramembranous ossification must be related solely to the development of bones and not to the development of bone. Bone formation always occurs by the differentiation of embryonic or adult connective tissue, a product of the primitive mesenchyme.[14]

In bone formation, two fundamental processes may be distinguished: the synthesis of a calcifiable protein matrix, and the deposition of mineral salts therein.

(b) *Formation of the organic matrix*, sometimes called osteoid tissue or osteoid, is an essential preliminary to the precipitation of inorganic salts in the formation of bone. Osteoid tissue is a product of the connective tissue cells during and after their transformation into osteoblasts. In the early embryo[20] osteoblasts arise by direct transformation of mesenchymal cells while in the older subject they are derived mainly from fibroblasts and reticular cells.[13]

It would appear that any primitive mesenchymal cell under the environmental influence which initiates osteogenesis may behave as an osteogenic cell.[20]

Custer[21] states that although with the maturation of organs and tissues the fixed mesen-

chymal cells fall into relative obscurity they retain their totipotential properties throughout life. These cells occur mainly in association with the delicate tissue framework of the bone marrow, spleen, and lymph nodes but they are also distributed as reticular cells throughout the body, especially in the adventitia of small vessels. It is evident, therefore, that there is a large and widely distributed source of cells both primitive and differentiated with an osteogenic potential.

Connective tissue in which future bone is to be formed becomes highly vascularised[22], and the cells are seen to be connected to one another by their processes.[13] The osteogenic cells then tend to enlarge and take on a more orderly arrangement, sometimes forming rows, and commence to elaborate ossein. There is little evidence to show how this collagenous deposit is formed and it has been suggested that it is "secreted by the cells and alternatively, that it forms as an ectoplasmic deposit or is spun off the molecular film at the surface of the cell.[23]" It would seem that there is no longer any doubt that calcification of bone is the result of enzyme action initiated by the osteoblasts and there is some evidence that the deposition of collagenous substances is also an enzyme reaction. For example, collagen formation around degenerating fibroblasts and odontoblasts in the dental pulp supports this view. In the guinea pig, removal of Vitamin C from the diet stops the normal deposition of collagenous matrix in the pulp chambers of the persistently growing teeth followed by the degeneration and death of the odontoblasts. If after the death of the odontoblasts ascorbic acid is restored to the diet, within the next day or two a deposit of apparently normal collagen matrix will appear surrounding the debris of the odontoblasts. It may be suggested that the cells contain some substance, possibly an enzyme which persists for a time after the death of the cell and which can partake in collagen formation when other factors are normal.[23]

Early collagen laid down by the osteogenic cells is in the form of coarse fibres which soon become grouped into large decussating bundles averaging 12 to 15 microns in diameter.[20] The bundles run in all directions forming a loose meshwork about the cells which lie in the semifluid intercellular cementing substance. At this time the cells give a basophilic reaction and have increased in size to become polyhedral while still retaining their numerous interconnecting processes. The next stage to be observed is a condensation[14] (hyalinization) of the semifluid cementing substance of the connective tissue, around and between the fibrils, leading to a masking of the embedded fibrils because the cementing substance and fibrils now have almost identical refractive indices. This condensation of the cementing substance may be a change[14] in composition actuated by the cells or there may be a replacement[13] by an entirely new substance elaborated by the connective tissue cells. Fish[23] asserts that the intercellular substance is a continuous collagenous matrix and that more collagen is laid down by the cells to cement the fibres together, but while this is indicated by the hyalinization of the organic matrix and masking of the fibrils the argyrophilic reaction of the fibrils and the argyrophobic reaction of parts of the matrix which contain no fibrils (capsule of the osteocytes) point against this hypothesis. The formation of osteoid tissue is now complete and it separates the entrapped cells which may now be identified as osteoblasts and the blood vessels in disorderly fashion so that in effect uncalcified primary bone exists. Osteoid tissue is very resistant to absorption, due probably to the presence of native proteins. Only after these proteins are denatured by cellular action and inorganic salts are deposited in the organic matrix is this substance readily absorbed.

3. Calcification.

The mechanism of deposition of inorganic salts in osteoid tissue is still incompletely understood. At least two factors, local and systemic, are involved. The systemic factor is concerned with the supply of minerals, conveyed by the blood and made available in the fluids of the body at the site of calcification. It is also related to the varying solubilities of the intermediate and final mineral constituents of bone. The local factor determines the concentrations of the various ions and the specific localization of the precipitated bone salts. It has been repeatedly emphasised that whatever part living cells may play in calcification the composition of the mineral substance depends on that of the liquid phase.

The biochemical mechanism by which bone salts are formed and deposited has been extensively studied since Robison[24] gave the first graphic description of a "bone enzyme" capable of hydrolysing certain phosphoric esters *in vitro*. He showed for the first time that the ossifying cartilage of young rats and rabbits contained a very active enzyme which rapidly hydrolysed hexose-monophosphoric ester and also glycerophosphoric ester with the production of free phosphoric acid. Robison demonstrated the presence of a similar enzyme in kidney tissue but found that other tissues examined, including non-ossifying cartilage from ribs and trachea, contain the enzyme in very much

smaller degree or not at all. At this time it was found that some ester of phosphoric acid is present in small amounts in plasma and in relatively large amounts in blood corpuscles. Robison[24] stated "if this ester is hydrolysable by the enzymes of ossifying cartilage the result would be an increase of $HPO_4^{=}$ ions in the cartilage." It was therefore suggested that this enzyme might play a significant role in the process of ossification. The enzyme was later referred to as "alkaline phosphatase." In an attempt to determine the site of activity of the phosphomonoesterase, bones from severely rachitic rats were split lengthwise and one half of each immersed in a solution of calcium hexosemonophosphate, the other half being immersed in normal saline. Both groups were incubated for two days at 37°C. After suitable preparation sections of these bones were made and stained with silver nitrite. The inorganic phosphate is converted to silver phosphate which is darkened by light. The half which had been incubated with saline showed the customary greatly enlarged zone of provisional calcification practically devoid of phosphate while the half bone which had been incubated with calcium hexosemonophosphate solution showed an intense black stain in the matrix of the cartilage cells at the epiphyseal junction when treated with silver nitrite. This could have been due to adsorption of the ester but it was noted that the deposit occurred only throughout the depth of the zone of provisional calcification and not in the proliferating cartilage as may have been expected if adsorption were the sole cause. Robison's theory of calcification may be briefly stated thus: A phosphomonoesterase is present at the site of calcification. This enzyme liberates phosphate ions from organic phosphates, thus producing a local increase in the concentration of phosphate ions. Consequently the solubility product of calcium phosphate is exceeded and the salt is precipitated at the site of enzyme action.

In 1930 Fell & Robison[25] published results of an investigation into the *in vitro* phosphatase activity of (a) cartilage which normally does not ossify (the distal end of Meckel's cartilage in the embryo fowl) (b) membrane bone and mandibular mesoderm from around Meckel's cartilage and (c) ossifying cartilage. All three tissues were obtained from the embryonic and post-embryonic mandibles of chicks. It was found that: (a) The distal end of Meckel's cartilage showed no sign of cell hypertrophy, did not ossify and did not synthesise phosphatase. (b) Mandibular mesoderm or membrane bone surrounding Meckel's cartilage, from the same embryos, gave very different results and showed that "membrane bone as well as cartilage bone synthesises phosphatase."[25] (c) Ossifying cartilage (palatoquadrates) showed a similar phosphatase activity.

Distribution of alkaline phosphatase in bone and cartilage.

It may be advantageous at this stage to give some details of a recent extensive survey of the distribution of alkaline phosphatase in bone and cartilage. In 1948 Greep, Fischer and Morse[26] described a process whereby it was possible to show histochemically the occurrence of phosphatase in adult calcified tissues. Hitherto the alkaline phosphatase had been destroyed during decalcification of the specimens. The ground substance of bone is devoid of alkaline phosphatase and in these preparations provided a colourless background for the phosphatase-laden, darkly staining Haversian canals and Volkmann's canals. The osteocytes were somewhat variable in their reaction, some without enzyme while others were weakly active. The osteoclasts showed little or no reaction. The periosteum and the endosteum showed moderate staining and clearly outlined the cortex of the shaft.

In highly organised centres of endochondral ossification the phosphatase distribution imparted a division of the epiphyseal plates into transversely located zones of varying width according to different local enzyme concentrations. The "zone of proliferation" contains no phosphatase in the interstitial substance and only a trace in the cells. Phosphatase appears in the cartilage cells as they enter the zone of hypertrophy and begin to enlarge and arrange themselves into columns. The cells were seen to become progressively richer in phosphatase as they move away from the epiphyseal plate. The enzyme is also seen in the matrix of the proximal third of the zone of hypertrophy and becomes more concentrated in the distal portions. In the zone of provisional calcification phosphatase is present only in the matrix. The capillary tufts from the marrow which invade and open up the cartilage columns and the associated osteoblasts are extremely rich in phosphatase. The osteoblasts showed varying content of the enzyme, those near areas of active calcification staining most intensely while osteoblasts located where bone formation is less rapid show a gradually diminishing concentration.

Articular cartilage shows an extreme variation in the local concentration of alkaline phosphatase. A broad band bordering on the articulating surface is absolutely devoid of enzyme activity. Beneath this clear zone the cells contain a great amount of phosphatase and the matrix also shows some signs of reaction. At the junction of cartilage and bone

over the entire circumference of the epiphysis of the young animals examined the osteoblasts are intensely phosphatase-positive.

In resting hyaline cartilage there is no trace of alkaline phosphatase in the interstitial substance and none in the cytoplasm of the cells, and the nuclei show a faint reaction in the nucleoli only.

In bone marrow the authors reported an uneven distribution and the identification of exact cell types was impossible.

The enzyme hypothesis of Robison was criticized and it was suggested that bone slices from near the epiphysis could be calcified by immersing these slices in solutions of inorganic salts provided that "the concentration of the bone-forming constituents and the reactions are nearly those of normal serum."[27] Robison and Soames[27] tested the validity of this statement by immersing slices from the femora and tibiae of rachitic rats in solutions containing constant concentrations of calcium and inorganic phosphate (10 and 3.5 mg. per 100 cc. respectively) but different concentrations of phosphoric ester.

> No calcification occurred in any of the slices immersed in the solution which contained no phosphoric ester. The addition of glycerophosphate, equivalent to only 0.5 mg. organic P per 100 cc. caused some calcification to occur, while the extent of the deposit was increased with each further increase in the amount of ester.[27]

Martland and Robison[28] in 1926 showed that apart from the store of hydrolysable phosphoric esters in the red corpuscles, such esters occur in plasma in amounts usually between 0.2 and 0.5 mg. P per 100 cc. but sometimes exceeding this amount.

A further experiment was performed in which the concentrations of calcium and inorganic phosphate as well as that of organic phosphorus were varied. It was found that calcification did not occur in the absence of organic P where the value of (Ca)x(P) was below 40 and only rarely at this level. It was concluded

> while calcification *in vitro* may occur in solutions of inorganic salts supersaturated with respect to the bone salt, calcification will take place with lower concentrations of calcium and inorganic phosphate if phosphoric ester is also present even in a very small amount.[27]

While carrying out an experiment on the effect of chloroform and other organic solvents on the phosphatase activity of calcifying bone slices Robison, McLeod and Rosenheim[29] found that bone slices, after treatment for 24 hours with organic solvents (chloroform, acetone, alcohol) or desiccation *in vacuo*, would subsequently calcify in solutions containing phosphoric ester. Similar slices would not calcify in inorganic solutions even of high (Ca)x(P) levels, while controls of untreated bone calcified in the inorganic solutions. Direct phosphatase tests were carried out on desiccated bone slices and it was shown that the enzyme activity was unimpaired by this treatment. Formaldehyde completely inhibited calcification in inorganic solutions and almost completely inactivated the phosphatase mechanism. Hence Robison *et alii*[29] postulated the operation of two mechanisms in calcifying hypertrophic cartilage:

> (a) the phosphatase mechanism which produces in the matrix fluid, by hydrolysis of phosphoric ester, a condition of supersaturation with respect to the bone phosphate, and (b) the second mechanism which favours the deposition of the bone salt from supersaturated inorganic solutions.[29]

This realisation of the presence of a "second mechanism" was the first indication of a deeper insight into the calcification of cartilage and bone. There was slowly evolving the idea that ossification was not merely the increase in local concentration of Ca^{++} and $PO_4^{\equiv}$ ions with resulting precipitation. Indeed, ossification implies complex tissue organisation governed by highly differentiated cells. At this stage no attempt had been made to explain why other tissues (including kidney, duodenal epithelium and gall bladder) which contain phosphatase do not normally calcify.

In 1932 Harris[30] examined the hypertrophic cells of cartilage for the presence of glycogen. He found that cartilage cells in the neighbourhood of the epiphysis store glycogen in direct proportion to their age. The more senescent the cell, the greater was the storage of glycogen. Osteoblasts and highly vascularised bone were found to contain no glycogen. It was suggested that the senescent or hypertrophic cartilage cells provided both the phosphatase enzyme of Robison, and glycogen, the latter for the production of the hexose moiety of a hexosephosphoric ester, this ester to provide a substrate for the action of phosphatase and thus an additional source of $PO_4^{\equiv}$ ions. The circulating body fluids provide both calcium and phosphates. The result is the deposition of calcium salts in the matrix of the senescent cartilage. Harris visualised the osteoblast as being able to control the re-absorption of mineral salt from the breakdown of calcified cartilage and the deposition and re-absorption of bone in this system. This was the first suggestion of a local source of phosphoric ester to augment the supply brought by the blood.

Until 1933 the method of estimating plasma phosphatase quantitatively had involved a lengthy and somewhat complex procedure. In that year Bodansky[31] devised a simple, accurate and rapid method for plasma phosphatase determination. He reduced the incubation period from two days to one hour, or less in the case of high phosphatase sera, and was able to use small quantities (1 cc. or less) of cen-

trifuged serum. A solution of sodium beta-glycerophosphate of known composition was used as the substrate. This method, with slight variation, is still in use today.

Further investigations into the process of calcification were reported by Robison and Rosenheim[32] in 1934. Evidence of the existence of the "second mechanism" required for calcification was becoming stronger but its nature was still completely unknown. It was apparent that the simple process of phosphatase causing an increase in the $PO_4^{\equiv}$ concentration in tissue fluid, already in equilibrium with the bone salt, was not sufficient to cause precipitation of the mineral part of bone. Phosphatase occurs in many tissues other than bone; if the enzyme in these other tissues sets free inorganic phosphate in a fluid already saturated with the calcium salt, why was this salt not deposited in the other tissues?

Robison and Rosenheim were able to demonstrate a great many differences between the function of the two mechanisms *in vitro*. The second mechanism was much more sensitive than the phosphatase to various agents including potassium cyanide, sodium fluoride, and desiccation. However the basic nature of the second mechanism remained obscure. They suggested that bone may not be entirely dependent upon blood for its supply of phosphoric esters, and agreed with Harris[30] that a cycle of synthesis and subsequent hydrolysis in the matrix could obviously serve as a mechanism for raising the level of inorganic phosphate at the site of calcification. Without giving details of a very long series of experiments to determine the nature of the "second mechanism" it may be said that the results obtained by Robison and Rosenheim emphasised the enzymic nature of this mechanism, and gave support to the suggestion of Harris.[30]

The inhibitory effect of sodium fluoride on calcification had been realised for a considerable time when Glock[33] attempted to determine the nature of this inhibitory effect. Robison and Rosenheim[32], using bone slices, had shown previously that 0.01 M NaF did not affect the hydrolytic activity of bone phosphatase, although 0.00001 M NaF inhibited the "second mechanism" of calcification. When phosphoric esters were present there was no inhibition. They though it possible that the "second mechanism" might involve a synthesis activity of the phosphatase of ossifying cartilage. They suggested that, in certain localities in ossifying tissue, the phosphatase might be so conditioned that its activities are mainly synthetic. This would cause a local increase in the concentration of phosphoric esters which thus serve as an increased potential concentration of inorganic phosphate.

Glock[33] saw the possibility of a relationship between sodium fluoride inhibition and glycogen synthesis. He determined the calcium and glycogen contents of the bones of two groups of rats at various stages of development. Both groups received the same daily ration of a stock diet; the first group had their daily ration moistened with 15 cc. water as the control, the other group receiving 15 cc. 0.05% NaF with the daily ration. The glycogen content and the degree of calcification in long bones and in the teeth of the rats were followed histologically and also the calcium and glycogen contents of bones were determined. The rats were killed at intervals from 4 days before birth up to seven weeks of age.

By reference to the results of chemical analysis it is seen that with normal rats the calcium content of the bones (expressed as percentage weight) increases steadily from birth, whereas the glycogen increases rapidly up to about 10 days and then rapidly falls to a somewhat constant level from the 26th to the 50th day. In the 'fluoride rats' calcification was inhibited during the first 15 days and then increased rapidly to the same level as in the control animals. The glycogen level was much lower than in the controls and reached a maximum at 15 days, at which time the degree of calcification suddenly began to rise. The author therefore suggested that the normal process of calcification in the early stages bears some relationship to the concentration of glycogen in the hypertrophic cartilage cells and that the delay in calcification observed in the fluoride rats might be attributed to their low glycogen content.

Histological examination of the bones showed that those obtained from foetal rats (5 to 4 days before birth) contained glycogen in the cartilage cells, particularly in the region which would later be the primary centre of ossification. At all stages and in both the primary and secondary centres the cells were seen to be heavily charged with glycogen just prior to calcification. Glock[33] stated

> It is thus apparent that the cartilage cells of both shaft and epiphyses contain quite a considerable amount of glycogen immediately before calcification begins, but once calcification has begun the glycogen falls off to a small and more or less constant amount.

The small, constant amount present after calcification begins is represented by that found in the hypertrophic cells adjoining the calcifying regions.

It was postulated by Glock[33] that the glycogen accumulating in the cells before calcification begins is the initial source of the phosphoric esters and that, after the initiation of calcification when the calcifying zone is very vascular, the blood affords another sup-

ply of these esters. This would also explain the fact that NaF only inhibits the early stages of calcification. Hence the inhibitory effect of NaF on the early stages of bone calcification could then be attributed to the low concentration of glycogen, which is at that time probably the source of the phosphoric esters. The subsequent recovery could be attributed to the fact that the blood, the later source of the phosphoric esters, is probably not significantly different in control and fluoride fed animals.

In 1943 Bourne[34] published the results of an extensive investigation into the distribution of alkaline phosphatase in various tissues. He analysed a large variety of tissues and found that those containing large amounts of phophatase included the superficial cells of the filiform papillae of the tongue, duodenal epithelium, submandibular salivary gland, bone marrow cells, endothelial cells of the vascular system and cells of the periosteum and endosteum. Tissues which contained little or no phosphatase included epithelial cells of the colon, and the cells of voluntary muscle. Bourne gave the results of an investigation into the distribution of phosphatase at various stages after boring a hole (1 mm. diameter) in both femora of guinea-pigs. He expected to find a general increase in the phosphatase reaction of all bones of the treated animals. In those killed 3 hours and 24 hours after the operation of boring the hole no difference could be seen in the phosphatase reaction of any of the bones compared with those of the controls. However, the time factor may not have been great enough or the injury too slight to provoke such a reaction. There was no immediate concentration of phosphatase in the clot filling the hole in the femur 3 hours after operation, nor any observable increase after 24 hours. By 24 hours there was an active migration of fibroblasts from the marrow and periosteum. These cells have a negative phosphatase reaction; however, some cells in the marrow of undetermined nature gave a positive reaction. Detailed observations were made of the distribution of phosphatase in the tissue filling the various holes in the femora after one week. The periosteum near the hole gave an intense reaction due chiefly to the deep staining of the osteoblasts and capillaries. Osteoblasts stained diffusely and cytoplasm and nucleus stained with equal intensity. Some cartilage had formed and when the cartilage cells were hypertrophied and had little matrix around then they often stained with great intensity. In some areas the cartilage cells were surrounded by matrix which gave a positive phosphatase reaction and the cells themselves seemed to contain very little of the enzyme but the cell capsule and surrounding matrix stained deeply. Bourne[34] suggested

The appearance was as if the cartilage cells (apparently derived either from a fibroblast or an osteoblast of the periosteum) had charged itself with phosphatase (probably by synthesis) and then as it produced and excreted the cartilage matrix it excreted the phosphatase with it.

Most of the fibres in the hole were found to give a positive phoshatase reaction and it was suggested that the fibres may have been formed by the "spinning" of a phosphatase impregnated film of protein from the surface of the fibroblast.[34] The cells of the capillaries gave a positive phosphatase reaction. It was also found that there was less phosphatase in regenerating and intact bone in scorbutic than in normal animals.

Gutman and Gutman[35] were attracted to the hypothesis that phosphoric esters formed during glycogen breakdown might serve as substrates for bone phosphatase. They quoted evidence to show that in liver and muscle the major pathway of glycogen degradation involves phosphorolysis and the formation of intermediate phosphoric esters. Thus the glycogenolytic cycle is initiated by the reaction:

$$\text{Glycogen} + \begin{matrix}\text{inorganic}\\ \text{phosphate}\end{matrix} \xrightleftharpoons{\text{phosphorylase}} \begin{matrix}\text{Glucose-1-}\\ \text{phosphate.}\end{matrix}$$

This enzyme had been shown to be present in liver, muscle and other tissues.

Gutman and Gutman[35] used the proximal and distal ends of the femora, tibiae and humeri of young rabbits for their experiments. They found that, when suitable concentrations of glycogen and inorganic phosphorus were added to ground epiphyses of young animals, the inorganic phosphorus content of the digest decreased rapidly with the formation of phosphoric esters. When whole epiphyses (including bone, marrow, blood, small-cell cartilage, etc.) were used the reaction proceeded unaided; however, when isolated hypertrophic cartilage from the epiphyses was used, the addition of muscle adenylic acid as a coenzyme was necessary to catalyse the reaction. The results showed the enzyme to be very rapid in action. This contrasted with the extremely slow reverse catalytic effect of bone phosphatase. Activation by adenylic acid further indicated that the results could not be wholly ascribed to bone phosphatase. The authors concluded that there appears to be present in calcifying cartilage a phosphorylative glycogenolytic enzyme system capable of synthesising potential substrate for bone phosphatase at zones of calcification, before blood sources become available.[35] They further suggested that glucose-1-phosphate appeared to be converted to other phosphoric esters too rapidly to serve as the chief substrate for bone phosphatase.

The results of further experiments on phosphorylase activity in cartilage were reported by Gutman, Warrick and Gutman[36] in 1942. They found that certain inhibitors, phlorizin and phloretin, completely or almost completely blocked calcification of cartilage *in vitro*. Adding glucose-1-phosphate to these solutions restored the capacity of cartilage to calcify well, even in the presence of phlorizin or phloretin. Since phlorizin and phloretin principally inhibit phosphorylase activity these results would imply that phosphorylase is not only present but that its action (formation of glucose-1-phosphate) is necessary for calcification of cartilage in a medium containing phosphorus as inorganic phosphate. Gutman *et alii* proposed that the process of dephosphorylation to provide $PO_4^{\equiv}$ for the bone salts depends upon the activities of three enzymes which they claimed to be present in calcifying cartilage: phosphorylase, phosphatase, and phosphoglucomutase. It was stressed that significant dephosphorylation of glucose-1-phosphate by phosphorylase was improbable because the glycogen stores of hypertrophic cartilage do not increase as bone salts are deposited, but disappear. The inhibition by fluoride, currently believed to prevent formation of phosphopyruvate from 2-phosphoglycerate by blocking the action of enolase, indicated that dephosphorylation by phosphatase did not take place prior to that stage in the cycle. It was claimed therefore that calcification of cartilage involves phosphorylative glycogenolysis at least to the point of phosphopyruvate formation. This system of enzymes was said to constitute part of Robison's "second mechanism of calcification".

While it was thus made clear that glycolysis plays a significant part in calcification, at least in the earlier stages, it would seem that the problem of bone formation is not thereby solved. While the supply of phosphoric esters by the breakdown of glycogen compounds may provide a source of $PO_4^{\equiv}$ ions it does not explain the familiar fact that calcium phosphate is not necessarily deposited from a supersaturated solution—the very difficulty that the "second mechanism" was proposed to clarify. Since phosphatase and phosphorylase are widely distributed throughout other tissues of the body, an explanation is still required for the factor which determines whether a tissue will calcify or not. After extensive investigation of the occurrence of glycogen in calcifying cartilage it was stated by Gutman[37] that the formation and rapid utilization of stores of intracellular glycogen would imply the presence within the cells of that tissue of the entire complement of enzymes now known to participate in the synthesis and phosphorolytic (or hydrolytic) degradation of glycogen.

In 1948 Slessor and Wyburn[38] tested a supposed osteogenetic property of phosphatase by inserting the enzyme (in concentrated solution in a calcium alginate gel, and also in pellet form) into the muscles of rabbits. The results were uniformly negative and in no case was bone formation observed.

In a concise report on the phosphomonoesterases Moog[39] suggested that it is likely that normal ossification involves some mechanism for the independent manipulation of calcium. This would explain the fact of the occasional ossification of phosphatase-free tissue. It would also explain the observation of Glock[33] that in fluoride-poisoned rats calcification appears to be deficient on histological examination but on chemical analysis they may be shown to have a calcium content equal to that of the controls. Moog has also drawn attention to the fact that calcification occurs in phosphatase-rich necrosed areas. In these cases the enzyme shows the same ability to deposit calcium phosphate as it does in areas of normal calcification. It was therefore suggested that the calcification problem should not only be thought of in terms of the peculiar properties of normally calcifying tissues, but also in terms of the restraint which cells of soft tissues appear able to exercise over their phosphate-liberating mechanisms. Moog concluded that alkaline phosphomonoesterase plays an essential role in the great majority of calcification whether normal or abnormal.

> The enzyme clearly supplies phosphate radicles for bone-salt formation, but an understanding of both the source of the substrate and the immediate fate of the released ions awaits a fuller clarification of the entire process the enzyme subserves.[3]

Little information has been available on the occurrence of acid phosphatase in the bone forming tissues. In an excellent review of the role of acid phosphatase in the body Moog[39] stated that acid phosphatase has a widespread distribution in human tissues and that the prostate gland is the richest source of this enzyme.* The widespread occurrence of another monoesterase with a pH optimum four units distant hardly simplifies the problem of assaying the physiological significance of the alkaline phosphomonoesterase. Adequate evidence was quoted to show that the two enzymes are mutually independent; the alkaline is a salt-soluble, the acid enzymes water-soluble. Histochemical tests have proved that the two enzymes sometimes occupy complementary locations in cells and tissues. It was pointed out that the alkaline phosphatase is the preferred enzyme of the cytoplasm, the acid of

*The serum of normal human adults contains about 2.0-3.5 Bodansky units (5-10 King-Armstrong units) of alkaline phosphatase per 100 ml. (Jaffe & Bodansky, 1943) and 0.6-2.0 King-Armstrong units of acid phosphate (Gutman & Gutman, 1938a).[39]

the nucleus. It was suggested that the alkaline phosphatase is more likely to subserve the functional activities of the tissue as a whole, while the acid phosphatase is more usually concerned with individual cellular metabolism. On the other hand, Moog[39] states unequivocally that both enzymes may be present in appreciable concentration in the cytoplasm of a single cell, e.g. in the cells of the connecting tabules of the kidney. This would appear to indicate a means of allowing two separate cellular activities to engage dephosphorylating steps simultaneously. An important, but as yet unsubstantiated, suggestion was made by this author:

> a possible implication of these data is that the matching of an active glycogen traffic with dephosphorylating mechanism operative in the acid range is one of the means by which the body prevents bone-salt deposition at unnatural sites.[39]

Experiments were reported by McKelvie and Mann[40] in 1948 on the role of alkaline phosphatase in bone regeneration around cancellous chip bone grafts in the tibiae of rabbits. Using the staining methods of Gomori and of Lorch the association of the enzyme with the cells concerned with bone formation was noted from the early stage of differentiation of the mesenchymal cell, through the osteoblastic stage, persisting to the formation of osteoid.

The following information on cell activity was provided by the results obtained on the sites of action of the enzyme:

(1) Presence of the enzyme indicates the region first influenced by the stimulus of osteogenesis.

(2) It identifies cells of mesenchymal origin destined to become osteoblasts.

(3) It provides an indication of the relative osteogenic value of the periosteum and the endosteum, and the part they play in repair.

(4) Its presence around bone transplants indicates their osteogenic potential, and clearly differentiates between old and new bone.

It was found that in the young animal the inner or cambium layer of periosteum possesses osteogenic properties; the cells of this layer show the presence of considerable amounts of alkaline phosphatase. These workers were readily able to trace the differentiation of the fibroblasts of the outer layers, where they may show a slight trace of enzyme, to the osteoblastic stage in which there is intranuclear, intracellular and extracellular alkaline phosphatase in considerable amounts. Particular emphasis was laid on the marked staining of the cell processes of the osteoblasts which link up with similar appendages from neighbouring cells. It was stated that these processes are undoubtedly associated with the formation of collagenous material which is the precursor of bone. This is in agreement with the suggestion made by Bourne[34], that the collagenous fibrils are formed by the osteoblast "spinning off" a phosphatase-impregnated protein from its surface. The process of endosteal bone formation and that associated with bone transplants were said to be essentially the same.

> The osteogenic cells of the endosteum are probably interstitial cells which undergo metaplasia and, in so doing, demonstrate the presence of the enzyme alkaline phosphatase. Cells of either the periosteum or the endosteum, which ever is nearer, may become associated with the formation of new bone around bony transplants.[40]

It was found that phosphatase could be demonstrated in the osteogenic cells up to the stage of mature osteoblast, but usually at no later stage.

> The young osteocyte may show small traces of alkaline phosphatase, but the enzyme is completely absent from the adult bone cell.[40]

For this reason, and from results quoted in the present review under "Origin and fate of the osteoblast", McKelvie and Mann gave the opinion that the enzyme alkaline phosphatase is closely linked with a cell which is concerned with the formation of the fibrous-like organic matrix of adult bone. According to results published by Moog[39] and Lorch[41] calcification can take place in the absence of phosphatase. McKelvie and Mann[40] also claimed that alkaline phosphatase occurs and can be demonstrated histologically under numerous conditions in which bone is not being formed. In these cases the enzyme is closely related to young fibroblasts and the collagenous intercellular material in both physiologic and pathologic conditions. In their own experiments these workers have demonstrated the presence of the enzyme in considerable quantities in periosteal fibrosarcoma (where there is never bone formation) in regions of polyostotic fibrous dysplasia (where there is an overproduction of fibrous tissue in the absence of calcification) and in the fibrous matrix in cases of Paget's disease.

The hypothesis was therefore advanced that

> the enzyme, alkaline phosphatase, is concerned more with the formation of the fibrocollagenous framework or matrix of the bone itself than with the impregnation of this framework with calcified salts.[40]

Conclusions.

It is evident that no system of calcification yet proposed explains the known facts as determined by experimental investigation of this process. It would also seem that the various workers have generalized too much in their approach to the subject without taking into account the differences which may exist be-

tween calcification at different sites, e.g. endochondral, endosteal, periosteal and soft-tissue calcification. This can doubtless be explained by the fact that the problem is indeed complex and as yet far from solution.

For normal calcification to occur in bone and cartilage adequate concentration of calcium and phosphorus is required in the enviroment. Deficiency of these elements in the body results in rickets or osteomalacia. Rickets may be produced in growing animals by suitable dietary measures, and on examination the histological picture is one showing large areas of uncalcified organic matrix. It has been shown that the fault lies at least partially in the chemical environment by the fact that the cartilage will calcify *in vitro* when exposed to suitable chemical solutions.[24, 27]

It has been established that glycogen accumulates in the maturing cartilage cells close to areas where calcification will occur.[30] The discovery that phosphatase[24] and phosphorylase[35] occur in the cartilage cells of the epiphysis and are closely associated with the process of calcification has induced extensive investigation of these substances and their possible influence on the mechanism of calcification. No definite conclusion has been reached on the role which these enzymes play in the calcification of tissues. It would seem from the results of recent work that alkaline phosphatase is mainly concerned with the formation of organic intercellular substance[40] rather than influencing the concentration of inorganic radicals. The possibility remains, however, that alkaline phosphatase is not a single enzyme but a system of enzymes capable of catalysing widely differing reactions. Again, there is the possibility that phosphorylase may be capable of catalysing reactions in both directions. Thus it could bring about the phosphorylation of glycogen compounds and under different conditions hydrolyse these compounds with the liberation of phosphate ions. The suggestion has been made that dephosphorylating mechanisms in soft tissues are carried out by an acid phosphatase and, by this method at low pH, calcification is avoided.

Howard[18] has considered the suggestions of previous workers that the matrix may take an important part in the mechanism of calcification. It has been insisted that the bone salts never undergo resorption without the concomitant disappearance of the matrix.[14] Follis[42] has pointed out that

wide osteoid surfaces (uncalcified bone matrix) are commonly seen in patients dying from chronic renal insufficiency, despite the fact that such patients have high serum phosphorus concentration and generally but little lowering of the serum calcium.

Again in experimental animals suffering from hypervitaminosis D where massive doses of the vitamin have been administered together with a low calcium intake, Muir[43] reports a hypercalcaemia along with a histological picture showing uncalcified osteoid surfaces. In these cases it was suggested that "the environment seemed suitable for calcification; perhaps the matrix was not."[18] Thus, in normal bone and cartilage matrices there may exist some characteristic property, which, in a reasonable environment, causes deposition of mineral salts, and which may actually determine the nature of the inorganic complex laid down. There is the further possibility that phosphate ions may be able to attach themselves to the matrix in loose chemical combination and thus build up a reserve against the time when calcification begins. This suggestion is supported by observations of extremely low Ca:P ratios in young bone, 0.35 and less.[44] More work will have to be done on the nature and evolution of the matrix before any definite conclusions can be drawn.

4. Bone Deposition.

Under normal conditions bone is constantly undergoing metaplasia, being formed in some places and destroyed in others. It should be emphasised that both processes occur simultaneously and that at any time one process may greatly exceed the other. Bone tissue has an enormous surface area and in cancellous bone the surfaces are greater per unit of bone than in compact bone. The surfaces may be arbitrarily divided into three types: those where bone is being formed, those where it is being resorbed, and those where no change is taking place. During the growth period of the individual and when repair of damage is occurring, bone deposition is the predominant mechanism.

Since all growth of bone is a surface phenomenon there must be cells present on bone surfaces which can form bone. All outer surfaces are covered by a layer of connective tissue containing cells with an osteogenic potential, and all inner ones are lined with a continuous layer of special cells. These cells are of two types, the less differentiated and more elongated osteogenic cells which have the capacity to become either chondroblasts or osteoblasts[9], and somewhat more differentiated and plumper osteoblasts which are the immediate precursors of osteocytes and matrix cells. Both osteogenic cells and osteoblasts can divide by mitosis and so increase their numbers. Moreover, osteogenic cells can readily differentiate into osteoblasts. Therefore when bone is required to be laid down in a certain area the number of surface cells can be increased to form more than one layer, by the division of osteoblasts, or the division and differentiation of osteogenic cells, or both. By this means the osteoblasts of the deeper layers

can lay down intercellular substance around themselves; thus a new layer of bone is added to the old surface and the new surface is still covered with a continuous layer of osteogenic cells or osteoblasts or both. This mechanism whereby bone grows is not, however, as simple as it first appears for the pattern of new bone must be so organised that adequate provision is made for the nutrition of the osteocytes. Hence, as described previously, no bone cell can be more than a fraction of a millimetre away from a capillary if it is to survive.

5. Bone Resorption.

Resorption is an integral part of the biology of bone and can be observed throughout the growth period of the skeleton. It is found in the adult whenever regenerative or reconstructing processes of bone occur. The growth of a bone, in contrast to the growth of bone tissue, requires that constant remodelling take place; thus bone resorption as well as appositional growth must occur.

> Bony structures are not malleable and hence the only way that the shape and size of any bone structure can be changed is by bone being added to, and taken away from, its various surfaces.[9]

It is not many years since terms like "creeping replacement"[11] to indicate a remodelling of bone, were seen in literature, and even today our knowledge of bone resorption is remarkably limited.

Weinmann and Sicher[14] claim that removal of bone always entails simultaneous disappearance of the organic and the inorganic components of the ground substance. By this, they probably mean that the removal of one part necessitates or causes the removal of the other and that the interval between the two processes is not great. This is indicated by the fact that they later state that decalcification of living bone does not take place and that resorption of bone probably starts with the removal of the organic components of the intercellular substance by a proteolytic action of the osteoclasts. The isolated calcium salts are then said to be carried away by the tissue fluids or by macrophages. In support of this statement Weinmann and Sicher report that "calcium salts have never been found in the osteoclasts but have been shown in macrophages even at some distance from the area of bone resorption."[14] The views of these authors regarding the nature of the stimulus for the differentiation of osteoclasts have been reported in an earlier part of this review. While the belief that osteoclasts are the prime cause of destruction of bone tissue has persisted for many years there has been no definite proof of this fact. The main evidence in support of the theory has been circumstantial, i.e., that large multinucleated giant cells (osteoclasts) are generally seen approximating an area of bone resorption. From the point of view of cause and effect this is not proof, for on the other hand resorption may be the cause of the presence of ostoeclasts. In describing the absorption of the terminal ends of the pubic bones in pregnant rats Ruth[45] states that osteoclasts were seen but not in impressive numbers considering the extent of the resorption. Again, McKelvie and Mann[40] reported experiments on bone transplants and stated with regard to the resorption of the transplanted bone:

> Osteoclasts are seen only for the first few days and it appears that they play no part at all in the removal of the bone transplants.

Results of some excellent experimental work together with very fine photomicrographs were published by Ham.[9] He described the surface of the osteoclast near to bone as appearing to have a striated border "as though covered with fine short cilia". It had been said earlier that these striated borders suggest that the erosive action of the osteoclast may be mechanical in nature at least to some extent. From his own experimental work Ham reported that thrice-frozen-and-thawed and untreated cancellous bone chips transplanted into muscle and left for four weeks showed a good deal of resorption and quite a number of osteoclasts. Some osteoclasts showed striated borders, others revealed none but striated borders were present on the bone surface from which the osteoclasts had shrunken. In some cases it was observed that the striated border was continuous between the osteoclast and the bone and in some of these

> it was possible, particularly with the phase microscope, to show that the cilia-like structures formerly believed to be parts of the osteoclast, were continuous with, and indeed were, the free ends of the collagenic fibres and fibrils of the organic intercellular substance of bone.[9]

Another pertinent fact was that the so-called striated borders of osteoclasts were only seen on bone surfaces where the collagenic fibres of the bone were disposed roughly at right angles to its surface. Further search of the histologic sections revealed striated borders on bone surfaces at which the fibres approached the surface roughly at right angles, even if no osteoclasts were present. The conclusion drawn was that the striated borders were nothing more than the fringed surfaces of bone at sites where bone was dissolving away. This suggested that the mineral phase of bone is contained chiefly in the mucopolysaccharide cement substance which lies between the collagenic fibrils and fibres of the intercellular substance and that, when bone is undergoing resorption at surfaces, the mineral containing cement is the first to be removed leaving a

narrow area of projecting collagenic fibrils. These fibrils in turn appear to be dissolved at their free ends at about the same rate as the mineral substance so that the fringed cilia-like border of the bone never becomes very thick.

The foregoing observations provide grounds for doubting if osteoclasts are so essential for, and potent in, bone resorption as has generally been believed. It is indeed possible that no cellular activity is required to bring about the resorption of bone. It may well be that certain cells are required at bone surfaces in contact with tissue fluid for the maintenance of the mineral content of bone.

So it could then be said that

the problem of preventing the salts of bone from dissolving into tissue fluid at surfaces is presumably solved by all bone surfaces, except where resorption is occurring, being covered (or lined) with a continuous layer of osteogenic cells and osteoblasts.[9]

Ham states that the covering and lining cells of bone can produce phosphatase and this suggests that they more or less continuously do so and so maintain an environment at the bone surface which favours deposition rather than solution of bone salts. Hence if bone surfaces are left naked to the tissue fluid or if cells other than osteogenic cells or osteoblasts directly abut on a bone surface resorption tends to occur.

If the latter hypothesis is correct then the role of osteoclasts is negative rather than positive and it would explain why resorption also occurs at naked surfaces or when other types of cells, such as tumour cells, are in direct contact with bone. Some evidence has been reported in favour of the different hypotheses on the mechanism of bone resorption and only a probable inference can be drawn from the experimental results.

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Esthetic Dentures and Their Phonetic Values*

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The purpose of this paper is to present a method of treating the problem of phonetics based upon the belief that nature knows best. Natural esthetics plays the dominant role but it is not the subject of choice; it is purely a subject of necessity. It is a subject of necessity because of the tremendous influence it exerts upon the primary subject, phonetics. Phonetics on the other hand is a subject that has long been neglected in dentistry. It is one upon which little has been written and less known. It is a subtle subject, the study of which we have hardly known how to approach, and this paper will give a new approach, and a new solution to this evasive phonetic problem.

This new approach is based upon the fact that it has been my observation and it is my opinion that phonetics is controlled 100 per cent. by esthetics. While studying and endeavouring to perfect phonetics it has been impossible for me to separate these two. In fact, I have found it necessary to study esthetics in much greater detail than ever before in order to help solve this problem of phonetics. But, in referring to esthetics, I do not mean the esthetics which are usually terminated by setting the correct teeth in their correct position in the arch. I mean the esthetics which are not terminated until the supporting structures of these teeth, that is, the denture base material, are accurately reproduced to the correct form of the interdental papillae. It is true that, if the teeth are not naturally positioned, one should not expect normal speech. But, even if the teeth are correctly placed, unless the true anatomy of the investing tissues which was lost incidental to the loss of the teeth is restored, clarity of voice and diction is impossible in dentures.

For a moment, let us consider ourselves a specialized group of architectural engineers, who are specializing in rebuilding the structures and acoustics of the oral cavity, engineers to whom I have the pleasure of pointing out that the natural architecture of the dental arches is the basis of acoustic and phonetic perfection.

These structures, these dental arches, were designed and built by nature, which we might well call the master architect. They were designed for the purpose of giving us three of the blessings of life: (1) the tools of mastication; (2) the benefits of an esthetic laugh and smile; and (3) the advantages in the refinements of singing and speaking.

We, as architectural engineers, specializing in the rebuilding of these oral structures after they have been destroyed by the inroads of age, infection, and extraction, must be prepared to restore not one or two but all three of these blessings. The simplest way we can accomplish this is for us to copy the natural basic architectural design as it has been given to us by nature. If we do this, we shall then be automatically reproducing in true position and form all the intricacies of these natural structures, not just the teeth alone, but also the balance of the architectural anatomy that has wasted away.

By this method, in addition to replacing the teeth, we shall be restoring the beautiful contours of the gingival and other tissues which are such an asset to an esthetic smile. Also, we shall be replacing in that mouth, which I call the oral voice box, in its true relation to the other oral structures, the same bulk of material which it contained originally. In this way we are assuring our patient of normal voice and diction. Finally, after all this, if we tint and colour the denture base material to more closely simulate living tissues, we shall then, and only then, have fulfilled the hope and confidence placed in us by our denture patients.

And while on the subject of tinting artificial gums, do you realize the American people spend millions of dollars each year to tint their fingernails—not to mention toenails? They spend many more millions tinting their hair the right—and the wrong—colour. And possibly the amount of money spent tinting complexions each year is comparable to a presidential budget. What do you think these same people would spend to have their artificial gums tinted to look like the real thing? The answer is self-evident, and yet this phase of prosthetic dentistry has been in the doldrums for years. The natural fashioning and tinting of our denture base material is almost a lost art, originally attempted only by those who made continuous gum dentures. Why is this? Any colour, tint, or shade can be developed in acrylic, and yet, since the introduction of the methyl methacrylate denture

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base material, we have relaxed and said, "This is it. Here is a material that really looks like the natural gums." Possibly we are not to be blamed since it was such a vast improvement over vulcanite and RESOVIN, yet the truth remains that we have made no attempt to put natural colours and character into our artificial gums. We have been asleep to the fact that we could and should be rendering our public a much finer esthetic service, and asleep to the fact that dentistry could be acquiring some of these millions that are being spent for tinting.

Esthetic Principles in Nature.

It is this belief in the error of our ways which has led me to practise and design dentures according to the principles of nature and not according to the principles of dentistry. I have found when this is done that dentures have an entirely new appearance and a naturalness of speech which is surprising. For this reason I often refer to them as "Dentures with a New Look—that speak—by themselves." In developing this "New Look" in dentures there are three phases to be considered:

1. Natural setting of teeth.
2. Development of an entirely new concept of denture form.
3. Reproduction of the natural colours of living tissues.

The natural setting of teeth involves many problems which, as we all know, are based on an understanding of sound fundamental principles and of studying patient individuality. However, there are four such principles which are neither taught nor recognized in dentistry today, and these we must observe if we expect to develop esthetic dentures that will speak by themselves. I have found all too frequently these principles are not incorporated in the setups on our modern dentures, and here is where the first of our subtle problems of phonetics enters the picture.

The first of these principles has a definite bearing upon esthetics and upon tooth-to-tongue relationship. It is that the natural plane of occlusion is parallel to the floor when the patient is standing or sitting in a normally relaxed position. One can easily recognize this fact by simply observing people. Many individuals, during conversation, show their teeth as far back as the first molar. It takes a careful observer but a short time to realize the accuracy of this statement. When the bite blocks are almost completed, it is well to have the patient stand and walk about, possibly observing something at eye level, and when apparently relaxed, ask him to hold steady, open his mouth slightly, and close on the thin blade of a cement spatula. Observe if the spatula handle is parallel to the floor; if it is tilted up or down, the bite blocks should be adjusted to bring it to a level plane. This procedure assures a normal display of teeth on either side of the mouth and places their occlusal surfaces, for the length of the denture, in correct relationship to the tongue.

The second principle is the development of the correct clearance between the anterior teeth. It has been found most advisable for the operator to first set up roughly the twelve anterior teeth and then contour the remaining bite blocks to allow adequate tongue space. At this point the first try-in should be made, and the final selection and setting of these twelve teeth is completed right at the chair. After these teeth are set, there should not be more or less than 1 or 2 mm. clearance between the incisal edges of the anterior teeth when the patient is unconsciously repeating the letter "S." This can easily be ascertained in the following manner: Have the patient count several times from 1 to 10. This count should then be speeded up, assuring the patient that speed is the only item of interest, until he is counting as rapidly as possible. When the patient really counts these numerals fast, as he hits the 6 and 7 in rapid succession, the lips automatically fly open and the true clearance can be observed. The teeth should then be adjusted so they are within the limits of 1 to 2 mm. clearance in this function. The only cases in which this clearance cannot be developed are those presenting retruded mandibles. In such cases the same procedure can be followed except the principal point to observe is that there should be only 1 mm. of clearance in the area of the cuspid and first bicuspid.

The third point of importance is to develop the correct amount of tongue room in the posterior part of the dentures. After the anterior teeth are finally set, draw a line on the lower bite block from the mesial of the lower cuspid back to the most lingual aspect of the retromolar pad area; another line is then drawn from this same point to the most buccal aspect of the retromolar pad area. The important thing is to keep the lingual surfaces of the lower molars within the confines of these two lines. (Fig. 1.)

Usually the position of the lower posterior teeth is controlled by the setting of the upper teeth. Normally the correct setting of the upper teeth will throw the lower lingual surfaces between these two lines but, if this does

not occur, the setting of the upper teeth should be adjusted so the lowers will fall within these limits. If it seems necessary to set the upper teeth too far off the upper ridge in order to accomplish this setting, then the lower teeth should be set up to a crossed bite, thus permitting a more normal setting of the uppers, and thereby keeping the lowers in a position of normalcy in their relation to the tongue and the retromolar pad. This principle can be easily established in our dentures and is one that has much to do with denture stabilization and natural tongue action.

Fig. 1.—Study model showing the lingual surfaces of the posterior teeth and their relationship to the retromolar pad area, and the mesial of the lower cuspid.

The fourth principle in the natural setting of teeth deals with the relationship of tooth to ridge. It is common practice in dentistry today to set teeth directly over the centre of the ridge. I believe this principle precludes the development of either esthetic or phonetic dentures simply because teeth did not originate from this position. It is common knowledge that postextraction tissue loss is greater on the buccal and labial aspects of the ridges than on the lingual. This creates a ridge centre which is definitely more lingual than was the centre of the teeth prior to extraction. This fact can easily be demonstrated by making cross sections of models through edentulous areas and comparing ridge centres with the position of the remaining teeth. (Figs. 2 and 3.)

Because of these observations and proceeding on the principle that teeth should be replaced in the exact areas from which they came, I seldom find it advisable to place any teeth over the ridge. They should be set in an anterior, buccal, and occlusal position, and it should never be necessary to grind properly selected teeth because of ridge interference.

Two reasons for the practice of setting teeth over the ridge are based upon theories of leverage and ridge absorption. The theory of leverage is questionable at the outset, but it is my belief that leverages, or the tilting of dentures, is more easily controlled by the balanced setting of teeth than by the positioning of teeth. In this respect one might follow the doctrine of Dr. T. V. Boswell of Springfield, Mo., who has been wearing full dentures for years. He sets teeth buccal to the ridge and makes the buccal slope of the buccal cusps of the lower molars the active inclined plane, in place of the lingual slope, thereby assuring a downward and inward drive on the lower denture during mastication.

The theory that ridge absorption is increased by setting teeth off the ridge may have merit; however, my personal experience does not substantiate this theory. Edentulous patients with a history of absorption or pyorrhea are treated as systemically undernourished individuals, and are routinely given medical physical examinations and nutritional advice. It is seldom necessary to refit healed cases. To consider sacrificing known esthetic and phonetic advantages, in favour of questionable results obtained by setting teeth over the ridge, would not be in accordance with patient histories.

Fig. 2.—Cross section through upper first molar area showing the normal tooth position on one side and the lingual position of the centre of the ridge on the other.

Fig. 3.—Cross section through the upper central incisor. Black lines indicate correct position of pin tooth in relation to a postextraction ridge. Arrow indicates lingual position of centre of ridge.

In the discussion of the natural setting of teeth the fact should be mentioned that there are, of course, those exceptional cases of individuals whose appearance and even speech can be improved by setting teeth differently from their normal dentition. Another point to consider is the fact that patients who are pleased with the esthetics and phonetics of their dentures seldom develop denture problems annoying to the dentist or themselves.

Fashioning the Denture Base Material.

The second phase of this subject, that is, the fashioning of the denture base material, is one which should be given detailed study. This is a neglected phase of dentistry, and our apathy toward this subject has created many of our phonetic problems. Words are completely inadequate to describe the differences that exist today between the form of our dentures and the form of those portions of the mouth they are supposed to reproduce. One reason we have not been conscious of this difference is that we have not compared them side by side. It behoves every one of us to procure study models of normally shaped mouths, some of the best dentures we can find, and then study and compare them. Using a magnifying glass, study in detail the form of the gingival tissues on all sides of the natural teeth, and then notice the unnatural way a denture is made. Study the shape and fullness of the bone and gum structures supporting these teeth, and then observe the dentures and notice how inadequately these structures are restored. Notice how we have been grooving our dentures and attempting to identify root prominences that do not exist. (Fig. 4.)

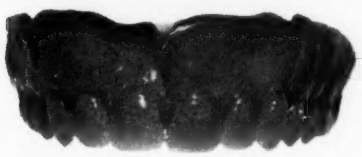

Fig. 4.—Denture showing natural gum contours on one side in contrast to the unnatural contours we have been using on the other side.

Also notice how crudely our dentures have simulated the natural palatal areas both with regard to the gross anatomy and the contours of the individual tissues. The ability we develop to reproduce nature accurately in our denture bases will control to a great extent the phonetic qualities we give our dentures.

Fashioning the palate areas is most important from the phonetic standpoint, and this study can be divided into three phases:

1. Contouring the lingual aspects of the porcelain teeth.

2. Duplication of any undistorted part of the palate of which there is a record.

3. Uniting these two areas in true anatomical form.

Properly contouring the lingual aspects of the anterior and posterior artificial teeth is seldom ever attempted in most dentures. Usually in waxing up cases, the palate form is started where the lingual porcelain ends. This is incorrect because it does not give the artificial teeth their full lingual length. This natural length should be developed in the wax and each tooth individualized, then the palate material develops with character from this point. A study of these two methods will show that in the latter there is much less bulk of material directly lingual to the teeth and this is a very important factor in pronouncing consonants. Improperly developed tongue space is the cause of many phonetic errors. This simple fundamental in developing tooth form has not been observed because we have not recognized this error as one of phonetic importance, and because we have been permitting inadequately trained technicians to do this work. The introduction of pinless and plastic anterior teeth has helped somewhat in the more natural fashioning of palatal areas. However, it should not be assumed that these lingual contours are always correct because many of them do not have a fully developed lingual surface and must be re-contoured in wax. If we do not care to carve our own dentures, we should at least respect the confidence our patients have placed in us and respect our dentistry sufficiently to see at least that this carving is done properly.

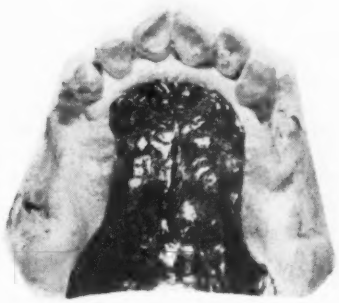

Fig. 5.—Tin foil adapted to the area of the palate that remains normal.

The second phase of palate reproduction is one that has to do with those portions of the palate which we are able to copy faithfully in every detail. The phonetic quality of dentures and their comfort to patients are in direct ratio to the amount of the oral voice box that we are able to copy exactly. This brings to attention another neglected phase of dentistry and another service that should be rendered more frequently than it is. It is the story of

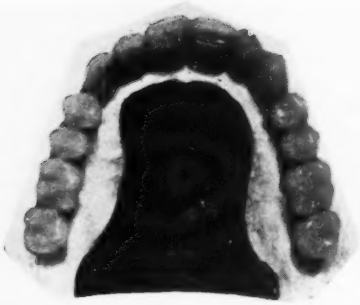

Fig. 6.—This model shows: (1) the extent to which palatal baseplate has been removed, (2) the lingual carvings around the porcelain teeth, and (3) the adaptation of the palatal wax.

study models. One of the first services that should be rendered every new adult patient is to make for that patient a permanent, and very accurate stone record of whatever teeth and investing tissues he may still possess, and to inform him that these models should be preserved as a permanent record. They may become of inestimable value to him in case of accident or the natural loss of teeth. Before making dentures, inquiry should be made of the patient as to the possibility of his having such models. Should he have, these may be used in copying palatal areas to much better advantage than just copying that small area of the palate which has maintained its true form during the years of periodic tooth loss and bone absorption. It is possible to visualize the reproduction of an entire palate from an old study model in which all the teeth are present, and the adaptation of it to a full denture for this same patient. Surely if his denture is contoured as his own palate was when the natural teeth were in place, it is going to function and feel better than one fashioned from memory.

In reproducing palates, it is most important to recognize how much or how little of the natural palate still remains. This should be outlined on the cast or on a former study model, if we are fortunate enough to have one. Then the following technique can be applied in order to develop the same details on the tongue side of the denture as exist or previously existed on the palate itself:

1. Outline on the cast the still normal areas to be reproduced.

2. Trim and carefully adapt with sponge rubber a piece of 0.003 tin foil to this outline. (Fig. 5.)

3. Remove the foil and set it aside for future use. (This step should be completed before the setup is started.)

4. After the setup is completed, remove the entire palatal baseplate.

5. Contour the lingual surfaces of all the teeth true to form. (Fig. 6.)

6. Carefully adapt one thickness of Kerr's red strip boxing wax to the outlined area without folds (by cutting). This wax will work without stretching at finger temperature and will assume the convolutions of the rugae, etc., quite easily.

7. The previously fashioned tin-foil control is now to be adapted to this wax. The wax, by virtue of its thickness, has created a dimensional change in the palate so that the tin foil is now slightly oversize. Carefully centre the foil and start matching it to the irregularities of the wax. As it is worked into place, because of its resiliency, it will collapse sufficiently to assume the new dimensional changes without losing its basic convolutions and the detail previously swedged into it. (Fig. 7.)

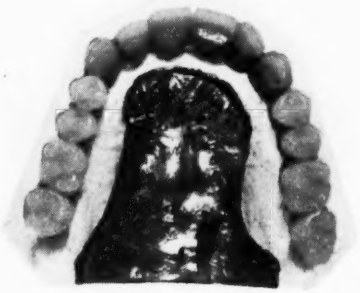

Fig. 7.—This shows the additional detail achieved by applying the previously adapted tin foil to the wax.

8. This foil remains in this position during the flasking operation, but is of course eliminated when the wax is boiled out of the case.

Two phases of the esthetic palate contour are now completed: the lingual of the teeth and a portion of the palate itself. The remaining portion must be waxed in next. (Fig. 8.)

It is here that a sound knowledge of tissue anatomy is imperative. Careful study of natural matured palate will develop this knowledge. Words are never adequate. However, there are several outstanding characteristics found in most palates which should be mentioned. For example, in the posterior area, that is, from the mesial of the first molar distally, there is a marked fullness lingual to the molars. It is essential to restore this full-

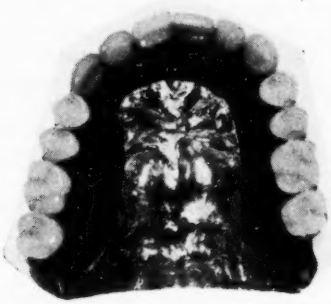

Fig. 8.—This shows how the balance of the palate should be waxed by hand to simulate natural palatal contours.

ness because the dorsum of the tongue is then able to control more easily the amount of air that escapes through the centre of the palate. To demonstrate this, simply count from 6 to 10 and observe the action of the dorsum of the tongue. Notice that it is more or less stationary on the lingual surfaces of the teeth and on this pad of tissue. The tongue elevates only slightly from time to time to control the amount of air it allows to enter the anterior third of the palate. Only the tip of the tongue moves rapidly as it whips the air about in the rugae area. If the lingual anatomy is not fully developed, the tongue is forced to spread itself in order to contact these surfaces, thus permitting too much air to enter the anterior section. This type of error is responsible for much of the whistling that occurs in dentures. The natural anatomy of the anterior portion of the palate is of great importance in pronouncing consonants. It is only in this area that rugae appear. I term it the playground of the tongue because 90 per cent. of the tongue's rapid manipulation in talking is restricted to this area and to a similar area lingual to the lower anterior teeth. These surfaces should be studied carefully to learn the different types of asymetrical rugae distribution and the usually convex contours of the gingival tissues. (Fig. 9.)

Fig. 9.—The average palate. Lines indicate the natural fullness lingual to the molars, the slight depression in bicuspid area, and average rugae position and type.

The buccal and labial aspects of dentures should next be considered. To realize the necessity for this study, compare the normal mature mouth with a collection of the finest dentures available. Where we ever conceived the contours we have been placing on these surfaces of our dentures is a source of surprise and astonishment to anyone making an honest appraisal of the situation. Such a comparative study will reveal the following points:

1. The interdental papillae on dentures are usually brought to a definite point with a flat or grooved effect above the point. The normal papillae are full-bodied and terminate in a blunt and even rounding tip. (See figure 10.)

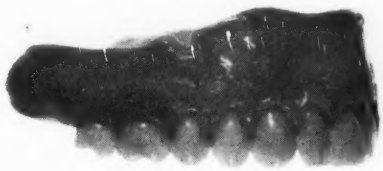

Fig. 10.—Denture showing natural anatomy such as interdental papillae, free appearance of gum margin, effect of stippling, and the full buccal contours.

2. The so-called free margin of the gum at the neck of the artificial teeth has anything but a free appearance. It is carved flat and tight to the tooth neck and then blended directly into the base material. In the mouth this tissue stands out in varying degrees, and it is drawn tightly about the tooth with a definite rolled edge. It simulates the cuticle around a fingernail, having a slight undercut so that it shows no direct attachment. This gum tissue never shows a stippled effect, but stippling is evident directly above this tense tissue and is often manifest in the body of the papillae. Tight or tense tissue and the flexible soft tissues are invariably free of the stippled effect. From an esthetic standpoint, artifical gums carved in this manner have a marked naturalness partly because the saliva adheres in this area more normally, enhancing the blend between the tooth and gingiva and dispersing the colours more harmoniously. (See figure 10.)

3. The area above the gingival tissues on dentures is either depressed, grooved, or carved out to simulate root prominences. It is either highly polished or stippled all over. It

seems generally to be contoured so as to eliminate bulk. The normal mouth will show almost the opposite. Only the tapering types will show very much in the way of root prominences. In the posterior area, all cases will have a marked fullness starting above the gingivae. This tissue is rounding, very firm, there is no stippling, and there are no grooves. Above this bulge there is often a depression composed of flexible tissue that blends itself with the muscle attachments. In the anterior area this fullness is usually not as marked but it does exist and slowly fades away into soft tissues and muscle attachments. (See figure 10.)

Another characteristic worthy of note and study is the consistency existing between types of cases and types of tissue anatomy. The gum contours of ovoid cases, for instance, are very plain. They seem to extend upward and outward in a smooth round fullness extending from one end of the arch to the other, and blending into the muscle attachments with no marked depressions or elevations. The square types are surprisingly similar to the ovoid except that they do exhibit stronger character and more interesting formations. The tapering types vary but in all instances they show stronger anatomic markings. Anterior root prominences are sometimes evident and the gingival tissues are beautifully developed.

Suffice it to say that from an esthetic standpoint the buccal and labial surfaces of dentures have been definitely inadequate, but from a phonetic standpoint they have been given absolutely no consideration. These surfaces may appear to have nothing to do with phonetics, but when it is realized that the natural surfaces, when reproduced on our dentures, create considerably more bulk, and this bulk, or the lack of it, has a definite influence on voice, then we must give credence to the fact that these areas are a part of our subtle problems of phonetics. Dentistry has not recognized the mouth as an oral voice box and that what we put in it can change its resonant qualities. In the past many of us have experienced difficulty in satisfying the demands of professional singers. I believe there are two basic reasons for this: (1) the artificial teeth were not placed in their natural positions; and (2) we did not restore the bulk of the tissue that had been destroyed in true anatomical position and form. In other words, to fill out naturally and replace the lost structures in the oral cavity is an appreciable factor in controlling the resonance of voice.

Webster defines voice as "Sound uttered by the mouth of living beings forming the basis of articulate speech." Hence, if the real basis of phonetic articulation is the voice, the better the voice, the better the articulation. Haven't we been putting the cart before the horse? In working on articulation and enunciation we have thought only of the teeth and the palatal surfaces of our dentures. Shouldn't we also consider the size and shape of dentures in voice and tone qualities, and build all denture surfaces as accurately as possible, in order to form the proper base upon which to set our teeth for more perfect articulation?

Phonetics is the science of articulate sound, and our modern age is replete with individuals whose livelihood depends upon perfection in this field. Today we have stage, screen, radio, television, and the clergy all demanding high quality in voice and diction. This demand we must be prepared to meet. We must recognize the fact that the answer is not just teeth and the setting of teeth, but also the development of naturally shaped dentures which will give the patient a better opportunity to utter normal sounds.

Tinting the Denture Base.

The last and possibly most dramatic phase of creating the "New Look" involves the tinting of the denture base material to resemble the tissues of the mouth. One cannot help but realize the inadequacy of present denture base material as a truly esthetic replacement after studying the many colours, shades, and tones that are found in these tissues. The basic colours and the intensity of colours vary with the type of individual, but the general shading of these base colours is fairly constant. As an example, the harder and more tense tissues are always lighter in shade than the softer tissues. The gingival roll and the cuspid eminence are usually lighter than their adjacent tissues, sometimes almost white. Deeper red or pink tones start in the body of the papillae and extend upward fanning out into the other soft tissues or disappearing in the lighter but harder prominences. Surprisingly strong colours exist in the region of the muscle attachments and other more flexible tissues. A study of palate colours, or possibly we should say a study of the lack of colour in the palate, may be of value to us when making dentures for stage personalities who must not exhibit foreign material in the palate. This important thing is that we must learn to observe and study these colours, and then do our best to reproduce them.

The first technical phase of tinting and shading a denture is to realize that the "New Look" must first be developed in the wax-up. This calls for careful study, particularly of the anatomic intricacies of the natural gingival structures and the various prominences that exist in the mouth which heretofore have not been incorporated in denture construction. In

attempting to restore the natural colour effects of living tissues we must remember that colour is dependent on form. Improperly fashioned dentures cannot reflect natural colour schemes, and disappointment will result if this phase of the work is neglected.

Much more time is required to prepare and wax up dentures for the reception of colour than actually to veneer the colours in place. The upper and lower cases must be waxed up in every detail just as the finished cases are to appear because practically no polishing can be done, or is required, after they are cured. An important phase of this detail is the required stippling. A simple way to place this stippled effect is to use a stiff toothbrush, removing all the bristles except the one tuft in the extreme end, and repeatedly stab this tuft into the wax in the desired areas. This leaves many tiny holes and flecks of wax on the high and low contours; by passing this part of the denture carefully through a low Bunsen flame, the flecks will disappear and the high spots on the wax will glaze over leaving the stippled effect where it is desired in the low spots.

After the wax-up is completed, the case is flasked in the usual manner except that a flashing of about one-fourth to one-half inch of a very dense stone is placed over the entire palate, teeth, buccal and labial surfaces of the denture. This is done before the upper half of the flask is invested. The finished denture can only be as smooth as the surface against which it is cured, so it is well to vacuum the stone and paint it on the wax.

When the flasks are separated and the wax eliminated, a separating medium that is colourless and that will not take away from the detail now showing in the stone should be applied. We have found many of the alginate tin foil substitutes suitable for this purpose. Tin foil and the heavier separators are unsuitable only because of the loss of detail incidental to their application.

The cases are now ready to be packed but, before the base material is prepared, the colours are laid in the flask wherever they are desired. The material used for this purpose has been made available to us by several manufacturers. They supply kits of various tissue-coloured and finely ground methyl methacrylate polymers which can be neatly tapped or shaken out of their dispensers. These different colours can in this way be dispersed and blended as the case requires. Usually a layer of about 1 mm. in this granular form is applied on the buccal or labial aspects over an area of four to six teeth at a time, and then sufficient monomer is applied by instruments or hypodermic syringe to satisfy the polymer. This monomer is applied at the edge of the polymer and permitted to seep into the polymer until it is saturated. The lighter shades are usually used in the first application, and this is followed by placing the other coluors and blends where needed. At no time is the loose polymer permitted to pile up over 1 or 2 mm. without an application of monomer. Correctly waxed-up cases permit the operator to identify and control the locations where the respective colours are to be placed. This work is continued in sections, because this is a simple way to control the loose powder and diversify the colour scheme. The palatal and other lingual surfaces can also be tinted and made to look very realistic after developing a knowledge of the colours that exist in these parts of the mouth. After the colouring is completed it should be given several additional applications of the monomer at 10 minute intervals. (Fig. 11.)

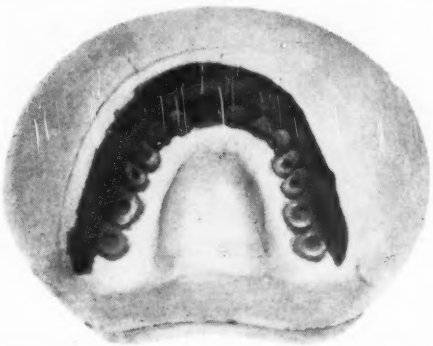

Fig. 11.—Flasked case showing the quarter inch of stone flashing about the teeth and the tinting colours applied to the labial and buccal surfaces.

The balance of the packing operation can be started immediately, or at any subsequent time. Care should be taken not to overpack or subject the case to severe pressures that might tend to rub the stains out of position. The injection method of packing is preferable but not essential.

After the case is cured and deflasked it is advisable to let the dentures soak as long as possible in a solution of sodium citrate. This will tend to break down the small particles of stone that lodge in the many crevices, making it easier to brush or pick out. Polishing is limited to the use of prophylactic brushes on the handpiece together with a very fine polishing agent. High-speed lathes and buffers create too much gloss on the dentures and tend to eliminate some of the fine detail that has been developed.

The procedure of carving and veneering colours on dentures by hand is naturally time-consuming and requires additional skill on the part of the operator. This restricts its use to those dentists who strive for the best results, and who are able to command an equitable fee. For this reason the esthetic values of this tech-

nique can hardly reach the general public. In an attempt to simplify this procedure, in collaboration with my ingenious technician and friend, Mr. Nelson Ingersoll, Jr., a technique was developed whereby these natural anatomic contours and colours could be fabricated by machine into a flexible acrylic veneer which can easily be adapted on denture surfaces during the packing operation. When available, these veneers may be procured by any dentist or technician and will be supplied in a variety of shades and sizes.

Their application will require no additional time or skill, and the surfaces to which they are applied will have a refreshing, natural effect both as to contour and colour.

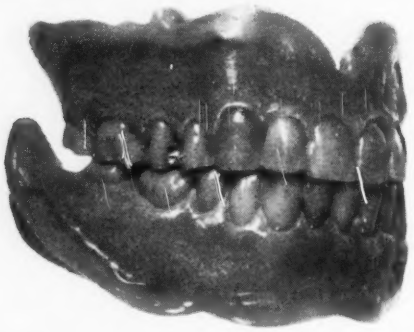

Fig. 12.—The "New Look" in action. Note the restoration of natural prominences, the full effect of the gingival tissues, and the colour diversification created by contours and stippling.

Conclusion.

In conclusion, it should be stated that the development of this "New Look in dentures —that speak—by themselves" can be a decided stimulus to esthetic and phonetic dentistry. (Fig. 12.)

Its principles are dictated by nature and are staring us in the face every day. The achievement of this result is based simply upon a determination to study nature with an open mind and then to give sufficiently of our personal time to apply this knowledge. The techniques suggested are within the reach of every dentist, and when the American public becomes conscious of this fact, plain acrylic dentures may become as obsolete as those made of vulcanite. It is hoped that others in our profession will also find that the application of these principles will bring immeasurable happiness to edentulous patients, and will remove from those contemplating replacements that fear of having to submit to an unnatural restoration affecting both speech and appearance.

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A Correlation Between Mellanby Hypoplasia and Dental Caries

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This preliminary paper is based on an investigation into the relationship between Mellanby Hypoplasia and the incidence of dental caries.

Mellanby Hypoplasia.

At the outset it must be clearly understood that we are not concerned with those gross enamel hypocalcifications and/or hypoplasias that are obvious to the naked eye. On the contrary the problem lies in the classification of the roughness of the enamel surface of teeth which clinically might be classed as normal.

Mellanby believes that the extent of this minute roughness is an indication of the degree of faulty calcification of the enamel; smooth enamel has excellent structure while rough enamel has poor structure. She has carried out several surveys which demonstrate that there is a close relationship between the structure of the tooth and its susceptibility to caries.

The enamel is divided into four groups depending on the roughness of its surface.

M-H0 is very smooth (as judged by passing a probe over the surface).

M-H1 is slightly rough.

M-H2 is moderately rough.

M-H3 is excessively rough.

Mellanby[1-3] showed that the presence of a liberal supply of Vitamin D (as well as a sufficiency of calcium and phosphorus) was the major factor in contributing to the formation of enamel of good structure. She went further than this and proposed that even after tooth formation was complete vitamin D would exert an inhibitory action on caries. These results were based on investigations using human material. The writer does not wish in this paper to discuss the controversy that has taken place concerning these hypotheses.

King[4] carried out a large scale dental survey of the Island of Lewis and reports that

> . . . an association of a fairly high degree has been found to exist between the quality of the surface structure of representative teeth of both dentitions and their susceptibility to decay.

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For many years, Mellanby, in association with other workers, has made similar observations.[5]

It must be borne in mind that the relation between Mellanby hypoplasia and caries may be partly due to the greater mechanical attachment afforded by the rougher surface for the plaque material.

Clinical Method.

The children used in this investigation were taken from those attending pre-schools in Canberra. They were aged 3 and 4. No attempt has been made to present the results of these two age groups separately because the small number of children involved would not permit it.

The average condition of the structure of the enamel surfaces of each child were divided into four groups: M-H0, M-H1, M-H2 and M-H3. The detection of these surface differences was carried out by passing a probe lightly over the smooth surfaces of the teeth. Care should be taken to ensure that the probe used is reasonably sharp, and that the sharpness is kept fairly constant throughout the investigation; otherwise the results will vary.

Practice is needed before the various degrees of roughness can be differentiated with any degree of accuracy.

Examination for caries was done by means of mirror and probe only. The results were expressed as the d.e.f. rate.

TABLE I

Average D.E.F. Rate Per Child in Each of The Four Enamel Roughness Groups

Enamel Classification	No. of Children Examined	d.e.f. per Child
H0	26	0.5
H1	71	3.7
H2	50	4.8
H3	23	7.2

Results.

These are summarised in Tables I and II.

The M-H0 group had an average of only 0.5 d.e.f. teeth per child, while at the other extreme the M-H3 group had 7.2 d.e.f. teeth per child. None of the M-H0 group had a d.e.f. of over 4, and 80% of them were caries free.

However, as might be expected there were some individual cases which did not conform with this general result. For example, in the group having the worst structure there were 22% who were caries free. This result will be briefly discussed below.

TABLE II

Percentage Frequency of Occurrence of D.E.F. Rates in Each of The Four Enamel Groups

Enamel Classification	d.e.f. Teeth			
	0	1-5	6-10	11-20
	%	%	%	%
H0	80	20	—	—
H1	36	40	14	10
H2	22	42	26	10
H3	22	26	17	35

Discussion.

In spite of the fact that Mellanby's hypotheses are not always accepted by the dental profession, it is clear that her observations on the relation between caries incidence and the surface structure of enamel are beyond argument.

However, even though the children belonging to the H3 group had the highest average d.e.f. rate, 22% of them were free of caries. In considering this phenomenon the power of the caries-initiating force must be taken into account. It can be assumed that factors such as a low sugar intake, good oral hygiene, good occlusion and tooth alignment will tend to reduce the prevalence of caries, irrespective of the basic susceptibility of the enamel. This is in keeping with a survey by Coumoulos and Mellanby[6] in which it was found that some children living in institutions had poor enamel structure and yet had low caries rates. This was probably due to the fact that the low refined carbohydrate intake reduced the tendency for caries initiation; hence the inherent caries susceptibility of the enamel did not have a chance to manifest itself. Thus the fact that enamel has been classified as M-H3 does not in itself mean that it must always be associated with a high decay rate.

Several methods have been used in attempts to assess the susceptibility to dental caries.[7] The lactobacillus count is the most usually used, but frequently it yields very uncertain results. The determination of the Mellanby hypoplasia would be more economical and more definite than the lactobacillus counts. A combination of both methods would probably be better, because it would take into consideration the resistance of the enamel as well as the force of the caries attack.

SUMMARY.

1. One hundred and seventy 3 and 4 year old children were examined for dental caries, and their enamel was classified as to the degree of roughness of the enamel surfaces.

2. A good correlation was found to exist between the two figures.

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The Problem of Prolonged Anaesthesia in the Dental Chair

Modern Methods for its Solution

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The recent advances in anaesthesia have rendered closer to fulfilment, not only the patient's, but also the dentist's and anaesthetist's requirements of a prolonged dental anaesthetic. In children general anaesthesia is particularly indicated when special problems in child behaviour confront the dental surgeon. Patients requiring specialised treatment fall into the following categories:—

1. Young children under four requiring extensive treatment.
2. Nervous, highly strung or unco-operative children over four years of age.
3. Spastic children whose motor paresis render them unable to properly co-operate.
4. Sub-normal children.

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Frequently these children require urgent treatment because the difficulty of coping with them has resulted in temporary fillings, inferior work, or the postponement of dental treatment until pain or septic mouth conditions force the issue. With spastic and sub-normal children the lack of oral hygiene usually contributes to the poor dental health. A carefully chosen modern anaesthetic technique enables the dental surgeon to provide a high standard of dental treatment for these children with a minimum loss of operating time and the avoidance of psychic trauma. Because of difficulty in handling these patients any attempt at normal methods of treatment must be rushed, and perforce technically unsatisfactory. The usual length of time required for adequate attention would inevitably result in severe emotional upset and a deepened fear complex.

The techniques used by the authors have been found suitable for hospitalised patients and also for ambulatory patients who are to be anaesthetised in the dental surgery.

When the anaesthetic is administered in the dental surgery the dentist has the advantage of familiar equipment and good operating conditions and the patient is saved the expense of hospitalisation. From the dental aspect provision must be made for good illumination of the mouth, a suitable mouth gag and an efficient sucker. A motor-driven aspirator is the most efficient, but one utilising water pressure from a surgery or workroom tap can be used. In the latter case, the water pressure available must be high to create sufficient suction.

The operative procedures can be expedited by re-arrangement of equipment and correct positioning of the patient's head. If the dental chair is swung on its base through 180°, the patient's head is then close to the bracket table and the electric motor, air syringe, mouth lamp and other dental unit instruments are conveniently at hand. A small pillow or rolled towel placed under the shoulders, or the nape of the neck, enables the head to be extended about 15° below a line through the long axis of the body. In most cases, this position affords good natural lighting of the mandibular arch, and the maxillary arch is illuminated by mouth lamp, or a mounted operating light, which can be readily trained on this area.

The use of an operating stool, and the ease with which the recumbent body may be raised or lowered, make for an ideal working position. The absence of saliva, good illumination and close proximity of all normal facilities enable any type of dental work to be carried out. While used most frequently for routine

fillings and extractions, this method provides the best possible conditions for more complicated techniques such as vital pulpotomy operation, or root canal therapy with immediate root resection.

The family doctor should be consulted about the child's general health and psychic make-up before deciding on any plan. The method of anaesthesia should be decided entirely by the specialist anaesthetist. The patient desires:—

1. A pleasant induction, with no sensation of suffocation or rotation.
2. If a child, no recollection of anything after swallowing capsules in waiting room.
3. No nausea or vomiting on recovery.

The dentist desires:—

1. Anaesthesia for from one half to two and a half hours.
2. No excessive bleeding.
3. No salivation so that the use of an angle mouth sucker becomes superfluous.
4. A quiet restful operating position preferably sitting down for long work.
5. A stationary target (so rare in children).
6. To be able to use cautery or naked flame with impunity.
7. The patient's rapid recovery to facilitate early ambulation.

The anaesthetist desires:—

1. A completely safe technique which is proof against any foreign body reaching the air passages even should the mouth be full of tooth fragments or blood.
2. A rapid return of protective cough and swallowing reflexes.
3. A non explosive technique.

It is emphasised that the methods described are the authors' approach to these problems, and do not represent the only ways in which they may be dealt with satisfactorily.

From an anaesthetic aspect dental cases divide themselves into:—

1. Children under the age of six years where the veins are usually difficult of puncture (in some cases children may be satisfactorily venipunctured down to the age of three years).
2. Children over six years old and adults where intravenous techniques are practicable.

For children under the age of six the method is basal narcosis followed by inhalational induction of anaesthesia. Trans-nasal endotracheal intubation is effected with maintenance of anaesthesia by nitrous oxide, oxygen and Trilene. On recovery from anaesthesia the child will recollect nothing beyond having some capsules in the waiting room, and will some time later wake up in its own bed, with its fifteen or so fillings completed in one sitting. Children are light enough in weight to be carried away by their parents and therefore may have the advantages of adequate premedication, to full basal narcotic level if necessary. This consists of giving orally half a grain of Nembutal per stone of body weight one hour before induction of anaesthesia, or rectally one gramme of Pentothal in 10% solution dissolved in tap water half an hour before induction. This latter method is very reliable but should not be used in children under one year of age. These children will not retain any foreign body in the rectum and furthermore are too young to require basal narcosis, anyhow. To be effective Atropine must be given in full, not homeopathic doses. This means from the age of two to five years 1/150 grain, thereafter 1/100 of a grain hypodermically. With these amounts there will be virtually no salivation whatsoever. In the very prolonged anaesthetic the Atropine effect may partially wear off; then, supplementary doses may be given intravenously with immediate effect. The authors feel strongly that the occasional atropine reaction of facial flush, tachycardia, etc., only occurs in the emotionally unstable child whose psyche has not been depressed by narcotic premedication before Atropinisation. After adequate premedication the sleeping child is gently transferred from the waiting room to the dental chair, which has been arranged as an operating table. Ethyl chloride, ether sequence is employed to rapidly take the child down to a plane of anaesthesia where the relaxation of the muscles of the jaw and throat is adequate to permit atraumatic intubation. Direct vision laryngoscopy is then performed, a well lubricated tube is passed nasally into the pharynx, and the tip caused to enter the trachea between the vocal chords. With the laryngoscope still in position the hypopharynx is packed without distension of the soft tissues. The packing gauze must be in the form of a continuous strip at least one inch wide and suitably moistened. Dry gauze can cause very severe pharyngitis.

Figure 1 shows the small gas reservoir bag situated at the patient's head directly attached to the nasal endotracheal tube. The reservoir bag serves the double purpose of an indicator of respiration and an adequate unrestricted

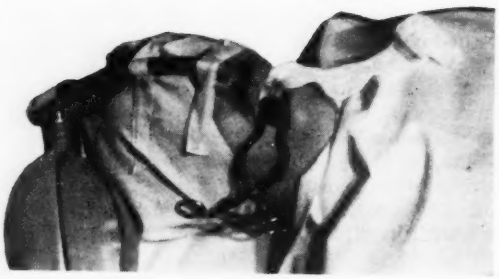

Fig. 1.

gas atmosphere to inspire from. By its situation close to the head carbon dioxide build-up is minimised.

Maintenance of anaesthesia from then on is entirely with nitrous oxide, oxygen and Trilene. Tranquil respiration, no salivation, and recovery of reflexes within minutes of cessation of anaesthesia are notable features of this technique. Vomiting, if it occurs at all, is limited to one transient "heave" just after the reflexes have returned. Nitrous oxide is chosen as the principal anaesthetic agent because it is readily reversible, does not stimulate mucous or saliva production, does not contribute to after-sickness, and in addition is non-explosive and non-inflammable. However, nitrous oxide being essentially a weak agent must be used in adequate concentration. Formerly this was only obtained with sub-oxygenation and the agent fell into disuse. With accurate calibration of the anaesthetic machine this concentration can be accurately administered. The authors' machine, in figure 2, is light (12 pounds) and permits the

Fig. 2.

administration of an accurate flow of nitrous oxide and oxygen to which absolutely minimal amounts of Trilene vapour are superadded. The Beckman diamagnetic oxygen analyser has been employed to check that the exact calculated concentration of gases is, in fact, being delivered to the patient. Since nitrous oxide and Trilene vapour are non-explosive, cautery, etc., may be used with impunity. However, for complete safety a period of ten minutes should elapse after intubation to make sure that all ether employed in induction of anaesthesia has been blown off from the patient's lungs.

For children over the age of six and adults, the maintenance of anaesthesia is essentially the same as has just been described, the only difference lies in the premedication and induction of the anaesthetic. Here, as one is dealing with a patient who is too heavy to be carried, basal narcosis or heavy premedication is avoided. Where maximum speed of recovery is essential, experience has convinced us that no premedication whatsoever should be given. The patient is sufficiently advanced mentally to permit a small prick in his arm without complaint after which complete oblivion ensues. No mask claustrophobia is present. The recently introduced muscle relaxing agents, either the short-acting decamethonium iodide or the ultra-short succinylcholine chloride solutions have facilitated intubation. Further, with their use minimal amounts of thiopentone only are required for induction with resultant rapid recovery.

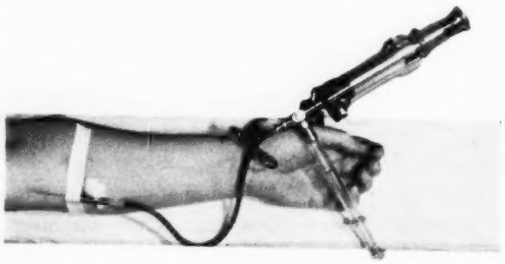

Fig. 3.

Figure 3 shows the intravenous technique employed with a two-way tap attached to the syringe allowing either relaxant, or thiopentone solution, to be delivered through a common tube to a vein in the anticubital fossa. A spring clip prevents reflux of blood into the needle and connecting tubing so that blocking of the needle cannot occur.

The techniques mentioned in this paper were elaborated at the United Dental Hospital, Sydney, and are also used by the authors in private practice. The procedure, so far, has proved eminently successful. The parents of the children have not been slow to comment on this innovation which allows all their dental work to be carried out in one sitting without psychic trauma.

SUMMARY.

The article describes the authors' approach to those patients requiring dental treatment where general anaesthesia is desirable or essential. A technique is offered embracing the use of modern agents which fulfils the desires of patient, dental surgeon and anaesthetist.

ACKNOWLEDGMENTS.

Thanks are due to the staff of the United Dental Hospital, Sydney, for their helpful co-operation, and to E. Slater, A.R.P.S., Clinical Photographer, Faculty of Dentistry, for providing the illustrations.

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The Biology of the Children of Hopewood House, Bowral, New South Wales

I. Observations on Dental Caries Extending Over Five Years (1947 to 1952).*

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Among the numerous facets of the unsolved problem of dental caries perhaps none has stirred more acrimonious debate than has diet. Unfortunately the word "diet" is often used loosely, and more often still it is confused with "nutrition". Admittedly diet and nutrition are closely linked, but they are by no means identical.

Many observations have been made (for example, Pickerill, 1912; Bunting, 1936; Colyer and Sprawson, 1938; Price, 1945; Mack and Urbach, 1948) on the relationship between diet and the incidence of dental caries, and it is generally accepted that no one particular diet is a *sine qua non* for the development and maintenance of sound teeth. Thus the Eskimo on a diet of protein and fat, the South American gaucho on a diet of protein, the South Sea Islander on a vegetable diet and the Australian aboriginal on a very mixed diet all enjoy relatively large measure of freedom from dental caries. A mixed diet is also used by so-called civilized peoples, and from time to time observations have been made that, even in those civilized communities where as a rule the prevalence[2] of dental caries approaches 100% (with a correspondingly high D.M.F.[3] value), it is possible so to modify the dietary pattern that the people living on such diets approach or even surpass in excellence the dental conditions of primitive peoples.

The object of this paper is to describe the condition of the teeth of a group of white children living under such controlled conditions in New South Wales.

Hopewood House and its Children.

The 81 children (boys and girls in practically equal numbers), who are the subject of this report, live in rural surroundings on the southern highlands of New South Wales at an elevation above sea level of approximately 2,000 feet. The living quarters are in what was formerly a country mansion and its spacious annexes; the buildings are set in 750 acres of undulating grassland. The staffing of the home is more than adequate, and earlier, when as infants the children required more attention, it was on an almost lavish scale.

The children now (February, 1952) range in age from four to nine years. The majority of them have been in the home since the earliest months of life. They are not a selected group, but are almost certainly what is genetically a heterogenous group of European stock.

The type of life led by the children is what is generally described as "healthy"; that is to say, they are well housed and well clothed, and have regular meals and regular exercise under supervision. Those of school age attend school; the younger ones attend a kindergarten within the home itself. As far as possible the children are made to feel that they are a large family living in what is virtually their own home.

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[1]We wish to acknowledge the assistance given by the National Health and Medical Research Council of Australia. This body has made grants for the renovation and operation of the mobile laboratory and for the purchase of equipment and supplies.

[2]Prevalence is used to indicate (i) the proportion of persons in a given population who are affected by the disease, or (ii) the number of D.M.F. teeth per child.

[3]D.M.F. value is the total number of decayed, missing and filled teeth. The expressions D.M.F. and D.F. are used in relation to the dentition present at the time of examination, whether or not this was wholly temporary or mixed temporary and permanent. Exfoliated temporary teeth have not been taken into account.

Observations.

Diet.

A study of any group of children living under controlled conditions is likely to prove interesting, but our interest in the Hopewood House group was considerably heightened by the fact that these children were "protected" from refined carbohydrates. We therefore made frequent visits to the kitchens, food-stores and dining rooms at meal times, and, although we have not kept a complete record, the diet consisted mainly of wholemeal bread, wholemeal biscuit, wholemeal porridge, wheat germ, fruits (fresh and dried), vegetables (cooked and raw), a small amount of meat[1], butter, cheese, eggs, milk and fruit juices. This was supplemented with vitamin concentrates, honey or molasses as a sweetening agent on occasion, and nuts (Youth Welfare Association of Australia, Annual Report, 1949).

As far as possible, food was taken uncooked and/or with a minimum of preparation, the idea being to present the food in its natural state. Notable for their absence from the diet were such items as sugar (white and brown), white flour products (including cakes and sweet biscuits) and any combination of these items. No tea was used. The water was drawn from the town supply. This water has been examined (Jones, 1949) for the presence of fluoride, but none was detected. We have not attempted to determine the fluoride content of the foods.

In order to maintain the kind of diet outlined above, those children who attended school were given at midday an appropriately prepared lunch before being allowed to mix with the other children. This was done with the aim of minimizing the danger of their exchanging items of food with other children.

When we began to study the children, attention was given primarily to dental caries. However, the scope of the observations is being broadened so as to obtain, for example, data on their general physical condition and on their haematological and biochemical condition. It is enough to state here in general terms that the physical status is (February, 1952) approximately the same as that found in a comparable group of the general population (Clements and Dowd, 1952).

Examination of Oral Structures.

When work began in February, 1947, the teeth were examined by one of us (B.L.) in the ordinary way with mirror and probe. On that occasion also plaster casts were made from the older children. It was not possible (at that time) to make radiographic examinations. In addition to the clinical examination, saliva was collected for bacteriological tests, with special reference to the occurrence of lactobacilli. The technique was similar to that described by Hadley (1933). The saliva was always collected in the early morning before any food had been eaten, and was transported rapidly to the laboratory in Sydney and examined within a few hours of its collection.

Subsequent examinations of the children's teeth have been made approximately twice a year, and from time to time bitewing x-ray pictures have been made to check the results obtained with mirror and probe. The carrying out of the various examinations has been considerably facilitated in recent years by the use of a mobile laboratory-*cum*-clinic.

[1]Practically no meat has been included in the diet since 1948-1949.

Results.

The results of the various examinations are set out below.

The figures are based on the results of the last series of examinations made. These were all completed in the period from September, 1951, to February, 1952. It is possible that on occasion there may have been a difference of some months between the last radiographic examination and the last clinical examination. However, in view of the extremely low rate of initiation of new lesions and the slow progress of established lesions, we feel that it is justifiable to group these results together.

Prevalence[1] of Dental Caries.[2]

Table I shows the distribution of the children and the prevalence of caries according to age and sex.

TABLE I

Distribution of Children and Prevalence of Caries According to Age and Sex

Age in Years	Number of Children		Children with Caries		Children without Caries	
	Boys	Girls	Boys	Girls	Boys	Girls
4	3	8	0	0	3	8
5	4	6	0	0	4	6
6	0	3	0	0	0	3
7	13	9	4	3	9	6
8	13	12	4	1	9	11
9	6	4	2	4	4	0
Totals ...	39	42	10	8	29	34
	81		18		63	

[1]See footnote (2), page 163.

[2]Pits and fissures were considered to be carious if the probe could be forced into them and remained fast. Smooth surfaces, such as approximal surfaces, were considered to be carious if the probe encountered a softened area or the radiograph revealed decalcification. No lesions were detected on the gingival third of labial and buccal surfaces.

Table II shows the prevalence of dental caries expressed in terms of D.F. values.

The distribution of the lesions shows a mode of 2.0.

TABLE II

PREVALENCE OF DENTAL CARIES (EXPRESSED AS D.F. VALUE) IN EACH OF THE 18 AFFECTED CHILDREN ACCORDING TO AGE AND SEX

Age in Years	Number of D.F. Teeth			
	Boys	Girls	D.F. Total	
7	1;[1] 1; 2; 3	3; 4; 6	20	47
8	3; 2; 5; 2	1	13	
9	2; 3	2; 1; 4; 2	14	

[1] This was a deciduous tooth and had been replaced by a permanent tooth at the time of the last examination. No other exfoliated deciduous tooth was carious.

Although the data presented above are concerned with the state of the teeth at the end of a five-year period, we have examined the children's teeth at intervals during this time. It has therefore been possible to observe the development of the lesions. Throughout the examinations, difficulty has been experienced in establishing with certainty the existence of some lesions. It was the practice firstly to examine the children's teeth with mirror and probe and then to examine the teeth radiologically; any possible lesion detected by either method was recorded. At the next examination particular attention was paid to those areas about whose condition doubt still remained, because of lack of proof from one or other of the methods of examination: radiographs were made at the time and were available to supplement the clinical examination.

Despite these precautions, certain areas remained in the category "doubtfully carious". Thus in six of the 18 children recorded as having caries the existence of lesions has not with certainty been established, and of the 47 D.F. teeth shown 20 were affected by conditions which belonged to the category of being "doubtfully carious". However, in Tables I and II any tooth which showed such a condition is included as being carious, and all children in whom it was not possible to establish with certainty the absence of caries are classified as having caries. Only three teeth developed lesions which involved more than one surface: these lesions were on a mesial surface and extended onto the occlusal surface. Six amalgam fillings had been inserted, three of them in first permanent molars, apparently by school dentists, as a prophylactic measure. These, with the exception of two doubtful lesions, were the only evidence of involvement of the permanent dentition. No lesion had advanced far enough to expose the pulp of the tooth. In view of these observations over the five-year period we feel that it is justifiable to state that the rate of development of lesions was very slow; indeed, in some teeth the disease may have been arrested.

Lactobacillus Counts.[1]

Lactobacillus counts were made at somewhat irregular intervals, but at least once each year, if the child was old enough to co-operate in the procedure. Table III shows the distribution of children without lactobacilli (that is, children who have never given evidence of the presence of lactobacilli) and Table IV the distribution of children with lactobacilli in their saliva (that is, children not certainly free of lactobacilli at the last examination).

The remaining 16 children not shown in Table III or IV were free from lactobacilli in their saliva at the last examination, but lactobacilli had been demonstrated in their saliva at some time previously.

TABLE III

CHILDREN WITHOUT LACTOBACILLI IN THEIR SALIVA AT ALL EXAMINATIONS: DISTRIBUTION ACCORDING TO AGE AND SEX

Children	Age in Years						Totals
	4	5	6	7	8	9	
Boys ...	2	2	0	2	2	2	10
Girls ...	8	6	1	1	4	0	20
Total	10	8	1	3	6	2	30

TABLE IV

CHILDREN NOT CERTAINLY FREE OF LACTOBACILLI IN THEIR SALIVA AT THE LAST EXAMINATION: DISTRIBUTION ACCORDING TO AGE AND SEX

Children	Age in Years						Totals
	4	5	6	7	8	9	
Boys ...	1	0	0	7	9	2	19
Girls ...	0	0	2	6	5	4	17
Total	1	0	2	13	14	6	36

[1] A lactobacillus count is the number of lactobacilli present per millilitre of a standard 10 millilitre sample of activated saliva. The sample, after measurement of its volume, is serially diluted in broth or peptone water and distributed in 0.1 millilitre quantities over a series of tomato-peptone-agar plates (Hadley, 1933). The colonies are counted after three or four days' incubation at 37°C., and the count is adjusted to represent the number of lactobacilli in a 10 millilitre sample of saliva.

TABLE V

DISTRIBUTION OF LACTOBACILLUS COUNTS ACCORDING TO MAGNITUDE

Children	Lactobacilli per Millilitre of Saliva				Totals
	1 to 10	11 to 100	101 to 1000	More than 1000	
Boys ...	4	9	2	4	19
Girls ...	6	7	2	2	17
Total	10	16	4	6	36

Table V shows the distribution of the counts according to their magnitude and according to the sex of the child. Thirty-six children are recorded as having lactobacilli, the counts ranging from less than 10 to more than 1,000. Of these 36 children there were eight in whom the nature of the organism so designated is somewhat doubtful. Only 28 of the 36 children therefore with certainty harboured lactobacilli. Of the 36, 26 had counts of 100 or less.

Association of Lactobacilli and Caries.

The association of lactobacilli and caries is shown in Tables VIA, VIB and VII, on the basis of which was calculated the χ^2 figure of 13.8 indicating statistical significance in the association ($P < 0.001$).

From Table V it appears that of the 36 children with lactobacilli in their saliva, 26 had 100 or less per millilitre. Of these 26, 20 were caries-free, whilst six were affected by caries to the extent of 14 cavities. This is for these 26 children an average D.F. value of 0.54 per child. Of six children with counts of more than 1,000 per millilitre, all had caries—20 cavities in all. This is for these six children an average D.F. value of 3.33 per child. These D.F. values are in the ratio of 1:6.

The three highest lactobacillus counts and the corresponding D.F. values are as follows: (i) 400,000 lactobacilli per millilitre, D.F. value six; (ii) 100,000 lactobacilli per millilitre, D.F. value two; (iii) 46,000 lactobacilli per millilitre, D.F. value four. The average D.F. value is four per child.

DISCUSSION.

Occurrence of Caries.

The figures given by Cameron (1953) are probably the best for purposes of comparison, because they are based on the examination of children resident in Sydney, New South Wales. He found that 96% of the children in the age-group four to nine years had dental caries and that the average number of D.M.F. teeth per child was approximately nine. Likewise an examination of groups of children aged from five to eight years in New Zealand revealed that 94.5% were affected, and that the average number of D.M.F. teeth per child in a group aged seven to nine years was approximately 11. Figures of the same order are available for the City of Brantford, Canada.

TABLE VIA

CARIES EXPERIENCE OF CHILDREN ACCORDING TO THE PRESENCE OF LACTOBACILLI DURING THE FIVE-YEAR PERIOD. (CARIES EXPERIENCE EXPRESSED AS D.F. VALUE OF EACH CHILD)[1]

Age in Years	Lactobacilli Present on												Total Number of Children
	No Occasion		One Occasion		Two Occasions		Three Occasions		Four Occasions		Five Occasions		
	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	Boys	Girls	
4	0;0	0;0;0;0;0;0;0;0	0	—	—	—	—	—	—	—	—	—	11
5	0;0	0;0;0;0;0;0;0	0;0	—	—	—	—	—	—	—	—	—	10
6	—	0	—	0	—	0	—	—	—	—	—	—	3
7	0;0	0	0;3;2;0;1;0	0;6	0;0	3;0;0;0	0	—	1;0	4;0	—	—	22
8	0;0	0;0;0;0	0;0;0;0;2;0	0;0;0;1;0	0;0	0;0;0	5	—	2;	—	3	—	25
9	3;0	—	0;2	—	0	2;1	—	4	—	2	0	—	10
Total number of children	10	20	17	8	5	10	2	1	3	3	2	0	81
	30		25		15		3		6		2		

[1] It should be noted that, owing to a variety of circumstances (for example age), not every child has been examined five times. Hence the younger children will tend to show less evidence of the presence of lactobacilli in addition to being less likely to have caries.

In three New Guinea villages the prevalence among children aged six to ten was 43%, 85% and 42% respectively, and the average number of D.M.F. teeth per child in the age group six to ten years was 1.64, 3.62 and 1.74 respectively.

The corresponding figures for Hopewood House are: prevalence 22.2% (18 out of 81) and 0.58 D.F. teeth.

Table VIII shows that the children of Hopewood House have approximately one-twentieth the amount of caries experienced by similar groups of white children in Australia and New Zealand, and that their freedom from dental caries is even greater than that enjoyed by the children of three villages in New Guinea.

The small size of the lesions deserves emphasis, and their slow rate of development is noteworthy.

Lactobacillus Count.

From Table III it can be seen that 30 out of the 81 children had never had any lactobacilli in their saliva, whereas (Table IV) 36 of the children have not been proven certainly free of lactobacilli at the last examination made. Frequently the numbers were very small, and Table V shows that 26 out of 36 children had counts of 100 or less, and only six of the 36 had counts of more than 1,000. It is generally considered (Hadley, 1933) that such low counts of 100 or less do not indicate susceptibility to the disease. Some authorities (Dewar, 1949, 1950) have even suggested that counts of less than 1,000 lactobacilli can be regarded as compatible with relative immunity to caries.

Association Between the Occurrence of Lactobacilli and Caries.

Table VII is a simple four-fold table which establishes statistical significance in the association between lactobacilli and caries. In the construction of the table, all possible lesions (certain and doubtful) have been included. Likewise, figures for the occurrence of lactobacilli include doubtful results. In adopting this procedure we have weighted the evidence against ourselves. Similarly no attempt has been made to exclude the low lactobacillus counts of 100 or less, despite the fact that such small numbers are frequently regarded as having no significance in relation to the existence of carious lesions.

Further, the figures given at the end of the section "Results" appear to indicate a quantitative relationship between the lactobacillus count and the D.F. value, when a group rather than an individual is being considered. The foregoing conclusion is drawn with a realization that the lesions are probably in their initial and active phases—there were no large, open cavities.

TABLE VIb

SUMMARY OF DATA PRESENTED IN TABLE VIa

Number of Occasions when Lactobacilli Detected	Proportion of Children with Caries
0	1 in 30
1	1 in 3.6
2	1 in 5.0
3	1 in 1.5
4	1 in 1.5
5	1 in 2

TABLE VII

RELATION AT THE LAST EXAMINATION OF THE PRESENCE OF LACTOBACILLI TO THE OCCURRENCE OF CARIES

Lactobacilli	Caries Present	Caries Absent	Totals
Present	15	21	36
Absent	3	41	44
Totals	18	62	80[1]

From these figures $\chi^2 = 13.8$ and $P < 0.001$.

[1]The disparity between totals 80 and 81 is due to the fact that complete data were not available for one of the 81 children.

Association Between Diet and Caries.

As was mentioned in the introduction, a number of authorities have drawn attention to the apparent association between diet and the development and maintenance of sound teeth. In the majority of cases the kind of diet compatible with sound teeth is one in which the food is eaten in as natural a form as possible—that is to say, with as little processing, refinement and cooking as possible. This is precisely the situation at Hopewood House. A brief description of the types of food eaten has been given earlier in the paper.

SUMMARY AND CONCLUSIONS.

1. In a population of children living under certain controlled conditions, 63 out of 81, whose ages range from four to nine years, were free from caries (Table I). The proportion of children without caries is far in excess of the proportion of children without caries in three New Guinea villages and in groups examined in Australia, New Zealand and Canada (Table VIII).

TABLE VIII

COMPARISON OF PREVALENCE OF CARIES AND OF D.M.F. VALUES FOR VARIOUS GROUPS OF CHILDREN IN APPROXIMATELY THE SAME AGE GROUPS

Place and Authority	Prevalences of Caries			D.M.F. Values		
	Age Group (Years)	Number of Children in Group	Percentage of Persons Affected	Age Group (Years)	Number of Children in Group	Average Number of D.M.F. Teeth per Child
Sydney, Australia (Cameron, 1953) ...	4 to 9	554	96.0	4 to 9	554	9.57
New Zealand (Hewat, 1948, 1952) ...	5 to 8	923	94.5	7 to 9	313	11.38
Hopewood House, Bowral, Australia	4 to 9	81	22.2	4 to 9	81	0.58
Brantford, Ontario, Canada (Hutton *et alii*, 1951)	5 to 9	2712	92.1	5 to 9	2712	8.00
New Guinea Villages (Sinclair *et alii*, 1950):—						
Patep II	6 to 10	14	42.9	6 to 10	14	1.64
Kavataria	6 to 10	13	84.6	6 to 10	13	3.62
Korovagi	6 to 10	19	42.1	6 to 10	19	1.74

2. The teeth of the children in this group of 81 are remarkable for the very small number of carious lesions. The average D.F. value per child (0.58) is lower than that for the children of approximately the same age group in three New Guinea villages (Table VIII).

3. No child had more than six lesions (Table II).

4. The rates of initiation of new lesions and of the progress of established lesions are very much below the rates in the general population.

5. Lactobacilli are present in the group and occur in some children as permanent components of the oral flora and in others apparently as transients. From one child with caries no lactobacilli could be isolated, and in 16 without caries lactobacilli have been detected on more than one occasion. Despite these discrepancies there is a statistically significant association between the occurrence of lactobacilli (irrespective of the count) and caries.

6. The outstanding difference in the environment as between this group of children and groups living in the population at large is in the nature of the diet. Foods containing refined carbohydrates (for example, sugar, white flour) are either excluded from the diet or eaten in very small amounts.

ACKNOWLEDGEMENTS.

We wish to thank especially Mr. L. O. Bailey, President and Founder of the Youth Welfare Association of Australia, under whose direction Hopewood House is conducted, and the staff of the home for their generous assistance in making possible the observations reported in this paper; also Dr. H. O. Lancaster, Honorary Consultant Statistician, who has kindly read the manuscript and checked the calculations. We also acknowledge assistance given in the laboratory by several members of the graduate and technical staff of the Institute of Dental Research.

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Dental Materials

*Current Notes No. (22)**

MOSCHELLANDSBERGITE—THE INSIDE STORY OF AMALGAM.

It is a strange story that we never have been quite sure as to what reactions take place in the setting of silver amalgam, our most frequently used dental material. It is also curious that what now appears to be the key to the mechanism has been with us all the time as the mineral with the imposing name of moschellandsbergite.

Since the clinical investigations of Black toward the latter part of last century many attempts have been made to establish the fundamental basis underlying his findings. First his "balanced" silver-tin alloy was shown to be essentially the intermetallic compound Ag_3Sn. The reactions that take place when mercury is added to this compound have proved less simple to discover. On this problem there have been two schools of thought associated with the names of Gayler and Troiano respectively. To discuss these in detail would involve diagrams and equations liberally sprinkled with Greek letters, but the two approaches may be pictured fairly in general terms. Both are in agreement in regard to the phases that are eventually formed. The point of departure concerns reactions in the early stages of setting, this being of most interest in accounting for the observed dimensional changes on setting.

Gayler concluded from her painstaking research that the intermediate phases first formed were a silver-mercury and a tin-mercury alloy, some of the former reacting with more mercury to give an additional silver-mercury phase. Troiano claimed that the phases first formed were alloys richer in mercury than proposed by Gayler. These then reacted with more of the original alloy finally producing the two silver-mercury and one tin-mercury phases suggested by Gayler.

Summing up, Gayler tended to envisage the amalgamation process as mercury gradually penetrating the silver-tin particles whereas Troiano was inclined to envisage a dissolution of the particles into the mercury; hence the intermediate phases in his system were correspondingly richer in mercury.

It has been stated that both workers agreed on the final products and to one of these, a silver-mercury phase, various formulae such as Ag_4Hg_5, Ag_3Hg_4 and Ag_5Hg_8 have been attributed from time to time. It now appears from x-ray diffraction studies that the compound is simply Ag_2Hg_3, identical in structure with the naturally-occurring amalgam moschellandsbergite. The formula Ag_2Hg_3 is intermediate between the other three mentioned and is actually closest to Ag_5Hg_8, the one most widely accepted until recently.

It is interesting to note that the relationship to moschellandsbergite was observed by two different groups of workers quite independently, apparently. In May, 1952, Frankel and Fankuchen[1] of the Polytechnic Institute of Brooklyn reported their x-ray diffraction studies which led them to conclude definitely that the amalgam filling is composed essentially of an intermetallic compound with the formula Ag_2Hg_3. In doing so, incidentally, they give an excellent and readable account of x-ray diffraction, what it is and how it is applied.

In March, 1952, Ryge, Moffett and Barkow of Marquette University presented a paper before the I.A.D.R. Dental Materials Group on the same topic. Their full report recently published[2] goes further and described not only the formation of Ag_2Hg_3 but also a crystalline tin-mercury phase identified as another of the end-products indicated by both Gayler and Troiano. Evidence for the third phase described by both earlier workers is not yet conclusive but it certainly does not appear in the early stages of setting as claimed by Gayler. By the same token no evidence was obtained for the intermediate mercury-rich tin-mercury phase claimed by Troiano.

The simplified picture of the setting of dental amalgams now in evidence should greatly assist in further elucidating the changes which take place in the setting of dental amalgam. It must be remembered, however, that in actual dental amalgam alloys there are complicating factors such as the presence of other elements besides silver and tin and also the physical form and condition of the particles.

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*Contribution from Commonwealth Bureau of Dental Standards.

Relation of Malnutrition to Fluoride Toxicity*

Discussions on the possible relationship of malnutrition to the incidence of mottled enamel have been frequent and varied in their conclusions. C.D.M. Day (Brit. Dent. J. 68, 409 (1940)) believed that his surveys in the Punjab province of Northern India indicated a higher incidence and a greater severity of mottling of the enamel among the malnourished inhabitants than among the well nourished individuals. Likewise, D. C. Wilson (Lancet 1: 211, 375 (1941)) stated that in her experience mottled teeth in children whose nutriture was assessed as subnormal tended to be accompanied by a pitting and attrition of the enamel which was not evident in the well nourished children.

However, in a survey in North and South Shields in England, where the drinking water supplies contained 0.25 and 1.4 parts per million (p.p.m.) of fluorides, respectively R. Weaver (Brit. Dent. J. 76, 29 (1944)) found no evidence of a relation between malnutrition and mottled enamel. Later, in a survey of a chronic endemic fluorosis area of Morocco, M. M. Murray and D. C. Wilson (Ibid. 84, 97 (1948)) concluded from studies of two groups of 25 children each, one European and the other Arab, that the Arab children had more severe mottling. The water supply was common to both groups, but the European children were judged to be much better nourished than the Arabian children.

M. Massler and I. Schour (J. Am. Dent. Assn. 44, 156 (1952)) conducted a survey in Italy in 1945 to determine if there were any possible effects of malnutrition on the response of the developing enamel to waterborne fluorides. Two Italian communities were selected for this survey: Campagnano di Roma, a community of about 1,000 inhabitants situated about 35 miles northwest of Rome, near the peak of Mt. Rossi, and Quarto, a community of less than 1,000 inhabitants lying in a valley about 15 miles from Naples, separated from the Mediterranean Sea by rocky hills.

The community of Campagnano di Roma was entirely agricultural and was well supplied with wheat, vegetables, and livestock. Throughout the war, the community was reasonably self-sufficient with respect to food. At the time of the survey, the inhabitants were moderately nourished on the average, but were not judged to be so well nourished as the average individual in most cities in the United States. Seventy-eight per cent. of the adults were judged to be in a state of adequate nutriture and only 20 per cent. were believed to be malnourished. However, only 32 per cent. of the children, 6 to 14 years old, were found to be well nourished and 50 per cent. were definitely malnourished.

The town of Quarto was entirely agricultural, but raised little besides apples. The economic status and the standard of living were low. Signs of malnutrition were apparent in nearly all the people. Evidence of frank rickets, dry scaly skin with multiple sores, angular cheilosis and red fissured tongues were found in almost every inhabitant by simple inspection. Only 1.5 per cent. of the children, 6 to 14 years of age, were judged to be well nourished, 2.9 per cent. were in a state of subnutrition, 39.7 per cent. were mildly malnourished, 44.1 per cent. were in a state of moderate malnutrition, and 11.8 per cent. were in a condition of severe malnutrition. The major nutritional disturbances in both towns were rickets, deficiencies in the vitamin B complex, in protein and in calories.

The water supply was obtained from a village well in both cases. The fluoride content of the water in Campagnano di Roma was 3.5 p.p.m. and at Quarto was 1.3 p.p.m. The amount of mottling of the inhabitants' teeth was scored from O for no mottling present to V for severe mottling. The amount of mottling in the teeth of the inhabitants of Campagnano di Roma was compared with the amount observed in Conway, South Carolina, where the fluoride content of the water is 4.0 p.p.m. The percentage of children with mottled teeth at Campagnano di Roma was 100 while the incidence in Conway was 88.2. In addition, the average extent or index of dental fluorosis was 2.52 for the children in Campagnano di Roma in contrast to 2.10 for the children of Conway. The amount of mottling of the teeth of citizens of Quarto was compared with the degree of fluorosis seen in Joliet, Illinois, where the water supply contains 1.3 p.p.m. of fluorides. The percentage of children with mottling in Quarto was 60.0, with only 25.3 per cent. of Joliet children exhibiting any signs of abnormality. The index of dental fluorosis in Quarto was 1.20 in comparison with 0.46 for Joliet. The inhabitants of Conway, South Carolina, and Joliet, Illinois, were reported to be in a much better state of nutrition on the average than the residents of the two Italian communities.

*Extract from Nutrition Reviews, Vol. 10(8): Aug., 1952.

The investigators commented in considerable detail on the differences in the amount of mottling between the inhabitants of the two American cities and those in the two Italian communities with comparable fluoride-bearing water supplies. They emphasized that the higher incidence of dental fluorosis among the Italian citizens could not be attributed to higher consumption of water; on the contrary, they felt that the water consumption in Campagnano di Roma and Quarto was appreciably less than in the United States Towns. They believed that the only discernible reason for the increased prevalence and increased severity of mottling was the fact that malnutrition was more severe in the Italian children and adults than in the United States. An observation which may strengthen this point is the higher incidence of moderately severe and severe mottling among the inhabitants of Quarto where the malnutrition was the most serious.

In an effort to study more exactly whether the degree of malnutrition might be correlated with the susceptibility to dental fluorisis, they determined the degree of association between understature and the extent of mottling and the degree of association between physical status as estimated by the Wetzel grid and the extent of mottling. In the first instance, the coefficient of association was 0.361, and in the second case it was 0.601, where perfect agreement would be 0.9 and no agreement would be zero. Thus, there appeared to be a tendency toward more mottling in the lower height-lower weight groups.

The investigators tended to implicate inadequate calcium nutriture as the important factor in the causation of the increased mottling rather than any of the other obvious deficiencies. This proposal was based largely on the fact that numerous studies with experimental animals have shown that calcium deficient animals were more susceptible to fluoride toxicity than their normal controls. The effects of other nutritional deficiencies on the toxicity of fluorides have not been studied in sufficient detail in experimental animals to permit evaluation of their potential relationships.

On the basis of present knowledge, the question of whether or not malnutrition influences the incidence and extent of dental fluorosis cannot be answered with any degree of certainty. Massler's and Schour's implication of calcium deficiency specifically seems to be premature, for a variety of other factors should first be considered in enough detail to be evaluated accurately. In the examination and evaluation of a survey such as the one described in this review, the reader cannot help asking questions about the constancy of the fluoride concentration in the water supply and whether or not the fluoride concentration of the foodstuffs was checked. For example, in the community of Quarto where the chief crop was apples, was a fluoride-containing spray employed for insect control without adequate cleansing of the apples before consumption? Again, were these communities sufficiently close to active volcanoes that volcanic ash containing fluorides might have been deposited on the vegetation of the gardens and farms in these areas? Likewise one must consider the question of water consumption which was dismissed as inconsequential because the investigators believed a low water consumption existed in these communities. Was the survey sufficiently detailed to determine this point with accuracy? Are the cooking procedures such in these Italian communities that more water is used in food preparation than in American towns, either because much water is needed to soak the uncooked foods, or because long cooking periods are used with extensive evaporation and hence concentration of the fluorides? None of these questions may be involved in the differences between the incidence and extent of tooth mottling in these Italian communities and towns in the United States with similar fluoride concentrations, but on the other hand, they cannot be casually dismissed as irrelevant.

Correspondence

"Silicate Cements"

Sir,

During the past few months the dental supply houses have received a large number of complaints regarding the prolonged setting time and, in some cases, complete failure to set, of silicate cements.

I have spent some time in discussion of this matter with a number of practitioners who have experienced this difficulty and suggestions which have helped them I wish to pass on to you as they may be of value to others. These suggestions concern two factors:

1. Freedom of the materials from impurities, and

2. The effect of temperature on setting time.

1. The manufacturers of silicate cements warn that these materials have a tendency to absorb the vapours of essential oils and that these oils prevent interaction between the cement powder and liquid, resulting in increase of setting time. For this reason they advise storage of silicate materials away from medicaments, particularly the volatile oils.

In manipulating the silicate cements it is most important that the glass mixing slab and any instruments used be absolutely free of impurities.

The setting of these materials is controlled by the chemical composition, and introduction of foreign materials will upset the composition, which is quite critical.

Discussion has shown that many practitioners use the same glass slab for mixing silicate cements as is used for mixing zinc phosphate cements, zinc-oxide eugenol bases and other materials.

Now, eugenol is an essential oil and is not soluble in hot or cold water. Traces of eugenol may be removed from a smooth glass slab by using an organic solvent or a sodium hydroxide solution, but it is very difficult to remove them entirely with soap and water.

Examination of glass slabs used in most surgeries will show that, after a short period of use, the surface of the glass is pitted and scratched and it would be difficult to remove traces of eugenol from the depth of these scratches even by the use of organic solvents. This is even more important in the colder months of the year as the traces of such volatile oils will not evaporate quite as readily as in the warmer periods.

For this reason it is recommended that a separate mixing slab be used for silicate cements exclusively and that it, and the instruments used in manipulating the cements, be stored where there is no danger of contamination by volatile oils.

2. Temperature is a factor in controlling the setting time of silicate cements and during the colder months it will be found helpful if the slab is warmed slightly before use. Standard texts recommend temperatures in the vicinity of 70°F. F. T. Pearson (*Dental Journal of Australia*, March, 1949, p. 138), recommends chilling of the glass slab before use. However, if the temperature of the slab is below the dew point of the room air, there is danger that surface condensation of water from the air will dilute the cement liquid and result in increased setting time. In addition to this, chilling of the slab may produce a temperature considerably below the optimum, tending further to prolong the setting time.

Several operators have found that application of these two suggestions has resulted in normal behaviour of silicate cement materials previously considered useless.

I trust that others may find them equally valuable.

Yours faithfully,
JOHN LEVETT.

43 Haldon Street,
Lakemba.

Honorary Editor: N. D. MARTIN, M.D.S.

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Editorial

United Kingdom Mission Endorses U.S. Evidence on Fluoridation

The problem of dental caries control is not only a major issue in the dental health of the community but it becomes important from a public health viewpoint. The observation, that a nutritional deficiency of fluoride contributed largely to dental caries susceptibility and that this deficiency is made up under some conditions by the natural fluoride content of certain drinking waters and can be remedied by the addition of fluoride compound to those waters lacking this element, is one of the most important findings in the sphere of dental caries control.

The major part of the investigation of the fluoride-caries relationship was carried out in the United States by H. Trendley Dean and his associates in the Public Health Service.

In England, however, these findings have been viewed with a certain amount of scepticism and disinterest but influenced by the accumulating mass of evidence in favour of fluoridation the British Ministry of Health sent a Mission to North America in February, 1952, to study all aspects of the fluoride-caries relationship. As a result of the Mission the findings on fluoridation have been published in a 100-page report.[1] In the conclusion of the Report many pertinent aspects of fluoridation are reaffirmed by this independent study. The relationship between the natural fluoride content of water and dental caries experience is substantiated and the evidence that the reduction in the incidence of caries which results from the use of fluoridated water is stated to be conclusive. It is further pointed out that there is no evidence to indicate that natural fluoride acts in any way or has any property different from artificially fluoridated water. This difference in the mode of action of fluoride is a common objection to the practice of fluoridation[2] but a great deal of investigation both in animals and human beings into fluoride metabolism and balance studying assimilation, excretion, retention and the physiological effect of fluoride in their various forms has always shown that they are active as the fluoride ion irrespective of its source.

The question of the toxicity of fluoride in water is also raised as an objection to fluoridation, but the United Kingdom Mission, impressed by the good health of people in both natural fluoride areas and those where fluoride projects have been in progress for as long as eight years, consider that the optimal concentration of fluoride necessary to decrease dental caries constitutes no danger to health.

The Report states:

> We have found no scientific evidence that there is any danger to health from the continued consumption of water containing fluoride in low concentration. In the areas where naturally occurring fluorides are present at a level of 1.0 p.p.m. mortality statistics do not indicate any hazard due to fluorides and medical experience in such areas has not produced any evidence of increased morbidity. Many suggestions have been made that certain ill-effects may nevertheless occur. We can only comment that the proving of a negative is extremely difficult. Meanwhile, we are impressed by the fact that millions of people are living in ordinary good health on waters containing fluorides at levels of 1 p.p.m. or more.[3]

The question of the varying concentrations of fluorides required in different climates has been recognised for a considerable number of years and it is frequently suggested that the difficulty of determining the exact concentration to be used in any particular area is a serious objection to fluoridation because of the risk of children consuming warm water and consequently having a higher fluoride intake, thus producing noticeable dental fluorosis.

Recent investigations relating temperature and humidity to water consumption, fluoride intake and dental fluorosis have been reported in the United States and provide a basis for the estimation of the optimal content of water under different climatic conditions.[4] It must be remembered, however, that the amount of fluoride required to give protection against decay under any condition is *always* less than that which will produce staining of the teeth.

The *British Medical Journal*[5], commenting on the Report, draws attention to the effects of diet on dental caries and states:

> Comparative studies show that much the greatest change in the incidence of dental caries is associated with an alteration of diet,

and further states that

> . . . . even the reduction in dental caries experienced by many communities under wartime conditions was greater than could be expected from drinking fluorine-containing water.

This statement is not supported by the comparison of Mellanby's[6, 7] findings on London children after the War—27% of five year olds were free of caries—and those of Ast and Chase[8] from the experimental fluoridation project at Newburgh, New York, where 56% of 5-year-old children were free of caries.

It is well recognised that the dietary control of caries is an effective measure, as the study at Hopewood House, Bowral, reported by Lilienthal and others in this Journal (page 163) has so adequately shown, but the mass control of caries by such a dietary regime is not practicable.

No one has ever suggested that the complete answer to caries control is provided by fluoride, for other factors are certainly involved in caries susceptibility, but the effectiveness and *practicability* of this measure

should compel those concerned with the great problem of caries control and those responsible for the dental health of the community to utilize every means for the practical realisation of caries control by fluoridation.

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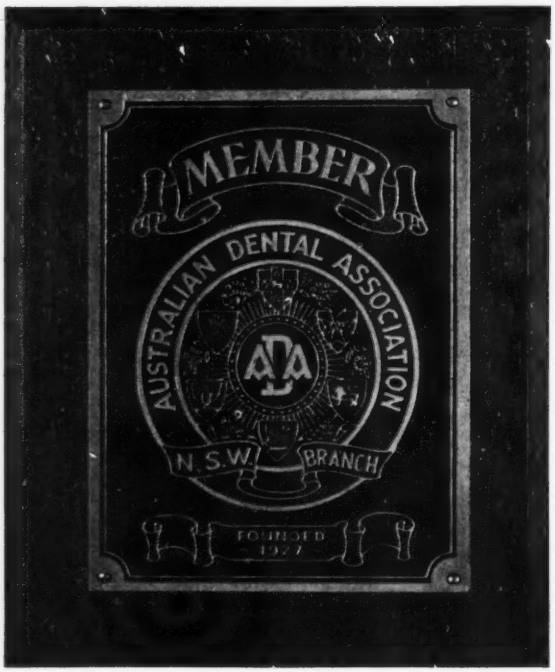
News and Notes

Membership Plaques



Members are advised that a limited supply of membership plaques is still available from the office of the Association.

The cost of these plaques illustrated above is 6s. 6d. per plaque.

British Dental Association

The British Dental Association has announced that, at the Extraordinary General Meeting of the Association held on 7th July, 1953, it was decided by special resolution that the annual subscription for each affiliated member of the Association would in future be £2/12/6 (sterling) per annum. This is to take effect as from the subscription due and payable on 1st January, 1954. Any member of the profession who has taken out affiliated membership with the British Dental Association is requested to note this fact.

Australian Society of Orthodontists

At a recent meeting of the Australian Society of Orthodontists held in Brisbane during the 13th Australian Dental Congress the Society elected the following officers:—

President: Dr. A. Thornton Taylor (Sydney).

Hon. Secretary: Mr. R. Y. Norton, M.D.S. (Sydney).

Hon. Treasurer: Dr. R. Halliday (Sydney).

Councillors: Dr. K. Adamson (Melbourne), Dr. D. F. Spring (Melbourne), Dr. V. P. Webb (Brisbane).

University of Southern California

Dr. William Ward Wainwright, noted dental research scientist and teacher, has received an appointment as professor of oral diagnosis and head of that department in the University of Southern California School of Dentistry.

A pioneer in the field of dental uses of atomic energy, Dr. Wainwright was a radiobiologist from 1947 to 1950 at the Los Alamos Scientific Laboratory in New Mexico while holding an appointment as professor of biochemistry and nutrition at the Washington University School of Dentistry in St. Louis and for the past three years he has been professor of radiology and head of that department in the University of Illinois College of Dentistry.

Flavelle Cup, 1953

The 25th Flavelle Golf Cup was played at The Lakes Golf Course on Thursday, 27th August, 1953. A field of 109 golfers took part but, unfortunately, rain prevented the Bowls from being played.

The following members of the profession were trophy winners:

Flavelle Cup:

Winner: K. Callen, square.
Runner-up: J. Newman, square.

Best Scratch Score:

I. Steele, 8 down.

Country Trophy:

K. Campbell, 4 down.

Handicaps:

12 and under: T. Royse-Smith, 4 down.
13-15: V. Golden, 2 down.
16-18: K. O. Binns, square.

4-*Ball Best Ball*:

Winners: J. Newman, J. Martin, 4 up.
Runners-up: E. McGovern, K. Callen, 3 up.

Association Activities

Australian Dental Association

FEDERAL NEWSLETTER No. 10.

Since the last Newsletter the Annual Meeting of the Federal Council of the Association has taken place in Brisbane on 3rd June, 1953. The Federal Executive has also met on 29th August, last.

Annual Meeting of the Federal Council.

The Annual Meeting of the Federal Council took place during the 13th Australian Dental Congress in Brisbane under the Chairmanship of the President of the Association, Dr. J. V. Hall Best, in the presence of the following officers:—

Dr. K. T. Adamson, Vice-president; Dr. J. M. Wark, Interstate Vice-president; Mr. N. E. Edney, Honorary Secretary; Dr. A. G. Rowell, Honorary Treasurer.

The Meeting was constituted by the following Delegates:—

Mr. W. A. Grainger and Brigadier H. McD. Finnie (New South Wales); Drs. A. Leigh Jones and R. Williams (Victoria); Drs. J. Luddy and F. R. Vincent (Queensland); Messrs. M. J. Barrett and B. Leach (South Australia); Messrs. K. F. Henderson and G. D. Henderson (West Australia); Dr. G. Hurburgh and Mr. N. E. Hecksher (Tasmania).

In attendance at the Meeting was Mr. E. F. Hewlett, Executive Secretary of the Association and Mr. A. H. Jackson, Secretary of the Queensland State Branch was an Observer.

The Minutes of the Annual Meeting of the Federal Council of 18th October, 1952, were confirmed and the *Report of the Honorary Treasurer* and an audited financial statement to the 15th April, 1953, adopted.

The Annual Report, the Report of the Standards Committee of the Association and the Report of the Association's representative as to dental matters dealt with by the National Health and Medical Research Council were adopted. A number of matters from these Reports which were discussed at the Meeting and on which subsequent action has been taken by the Federal Executive, are reported later in this Newsletter.

The Federal Council made the following appointments:—

Standards Committee: The personnel of the existing Standards Committee, namely:—Dr. Edward Marks, Professor H. K. Worner, Professor C. H. Down, Dr. A. R. T. Greenwood, Mr. Edward Campbell, were reappointed and the Council expressed their appreciation of the services of this Committee.

Representative Flying Doctor Service: Dr. W. S. Wilkinson was appointed as representative of this Association at the Annual Meeting of the Flying Doctor Service.

Election of Auditor: The Federal Council elected as Auditors of the Association for the financial year ending 31st August, 1954, Messrs. C. O Beck & Wayland of Sydney at a fee of twenty-five guineas.

Executive Secretary: The Federal Council confirmed the appointment by the Federal Executive of Mr. E. F. Hewlett as Executive Secretary until the next Annual Meeting of the Federal Council.

Special Business.

14th Australian Dental Congress: The Federal Council, under Clause 16(d) of the Constitution delegated to the Victorian State Branch the duty of conducting the 14th Australian Dental Congress. Victoria indicated that it was proposed to hold the 14th Australian Dental Congress within the period January/June, 1956, the actual date to be advised later, and that it was proposed that the Congress should be held within the precincts of the University of Melbourne.

National Dental Journal: A notice of Motion by the Queensland State Branch as follows:

"That a National Dental Journal be inaugurated."

was before the meeting.

The President stated that this Association was perhaps the only national dental association without a national journal. Discussion took place on the previous investigation made in this matter in 1945 and 1949.

Queensland then read to the Meeting a Memorandum on the establishment of a national journal prepared by that Branch in support of their motion. The President congratulated the Queensland Delegates on the comprehensiveness of the Memorandum and indicated that it would be circulated to all State Branches for their consideration. Queensland then withdrew the circulated motion, and after discussion on the advisability of calling a special meeting of the Council to consider recommendations, it was *Resolved*

> " That the possibility of establishing a national dental journal be investigated and, in order to further this matter, the Memorandum tabled by the Queensland Branch be circulated amongst the State Branches, following which a special meeting of the Federal Council be called at the discretion of the Federal President."

Repatriation Dental Treatment.

At the Annual Meeting of the Federal Council is was noted that, to that date, no advertisements calling for applications for Local Repatriation Dental Officers or for Senior State Repatriation Dental Officers had been issued by the Repatriation Commission.

Several delegates indicated that some confusion existed in some States as to the effective date of the new Schedule of Treatment and Fees.

At the Annual Meeting of the Federated Council the following resolution was passed:

> "That a letter be forwarded to the Repatriation Commission pointing out that several States had reported to this Annual Meeting that the Scale was already in operation; that the Federal Office had not as yet been officially notified of the commencement of operation of the Scale and asking whether the official notification might have gone astray."

Our concern over the delay in implementing the Repatriation Dental Scheme has been conveyed to the Repatriation Commission.

The Federal Executive is pleased to report that the Repatriation Commission has very recently taken initial action to implement this Scheme.

A broad outline, which includes the matter of eligibility, provided by the Commission has been forwarded to all State Branches for the information of their members.

The Commission have further advised that they propose, in the near future, to issue appropriate advertisements calling for applications for appointment as Local Repatriation Dental Officers and Senior Visiting Repatriation Dental Officers for each State.

Pharmaceutical Benefits.

At the Annual Meeting of the Federal Council the President reported that, during an interview with Sir Earle Page, the Federal Minister for Health, it had become apparent that certain difficulties foreseen by this Association in the extension to dentists of the right to prescribe under the Pharmaceutical Benefits Act had become concrete.

In view of the desire of State Branches to pursue this matter it was agreed that State Branches could individually undertake further investigations.

13th Australian Dental Congress.

The 13th Australian Dental Congress was a magnificent success and the Association is indebted to the Congress Commission and the Queensland State Branch for this outstanding achievement under the leadership of Professor S. Lumb, the President of the Congress.

Proposed Visit to Australia by Dr. Basil Bibby.

The Federal President wishes to report that, with the full support of the Federal Executive, negotiations have been finalised for a short visit to Australia by Professor Basil G. Bibby, D.M.D., Ph.D., who is Professor of Dentistry at the University of Rochester, New York, and is actively engaged in the affairs of the Eastman Dental Dispensary.

Dr. Bibby is one of the outstanding world leaders in dental research and preventive dentistry and should be visiting this country in the latter part of November. The Association is indebted to the United States Educational Foundation in Australia and New Zealand for this privilege.

In view of the responsibility of the Association to foster plans for the dental welfare of the community, an opportunity of consultation and discussion with Professor Bibby as to the latest trends and developments in preventive dentistry and research will be of incalculable benefit to the Association and scientific authorities dealing with these problems.

Federation Dentaire Internationale.

The drive for "supporting members" by the Federation Dentaire Internationale has been assisted by this Association and Dr. A. G. Rowell, who has been appointed National Treasurer for the Federation in Australia, has given attention to this matter.

The Annual Meeting of the Federation was held in July, 1953, at Oslo. The Association regretted that it was not able to appoint an Observer to this meeting.

Preliminary indication has been received that the 12th International Dental Congress will be held in Rome in 1956.

Fluoridation of Drinking Waters.

The National Health & Medical Research Council are giving further consideration to the question of fluoridation of drinking waters and have referred certain matters concerning this procedure to the Dental Advisory Committee which has been recently appointed to assist the Council.

The Federal Office is in receipt of the report by the United Kingdom Mission to North America to investigate fluoridation of drinking waters.

This Report is in the form of a British Government White Paper of 100 pages and contains an extensive analysis confirming the value of the fluoridation projects set up in North America.

Conference of State Branch Secretaries.

During the 13th Australian Dental Congress in Brisbane, at the instigation of the Federal Executive, a conference of State Branch Secretaries, consisting of two sessions, was held and proved most valuable.

Proceedings of this conference are to be forwarded to all State Branch offices in due course.

Commonwealth Medical Benefits.

Members are advised that the Federal Executive has given extensive consideration to the matter of Commonwealth Medical Benefits as they affect dental practice.

Federal Office, Sydney.
14th October, 1953.

J. V. Hall Best,
Federal President.

Australian Dental Association (New South Wales Branch)

GENERAL MEETINGS.

Annie Praed Lecture for 1953.

The Ordinary General Meeting of the Association on Tuesday, 22nd September, was the occasion of the Annie Praed Lecture for 1953. Professor A. J. Arnott, Dean of the Faculty of Dentistry within the University of Sydney, was the lecturer for the evening and, in opening his lecture, paid tribute to the memory of Dr. Annie Praed, touching briefly on her distinguished professional career and her services to the profession.

Professor Arnott had selected as his subject for this lecture "A Discussion on Problems in Dental Surgery Referred by Colleagues in the Dental Profession." The lecture, which was most adequately illustrated, was much appreciated by the large audience attending.

August General Meeting.

The Ordinary General Meeting of the Association of 25th August, 1953, was the occasion of a joint lecture by three lecturers: Mr. S. Levine, Mr. G. Morse Withycombe and Mr. R. Tompson, who presented to the members various aspects of the subject entitled "Periodontal Disease—A Practical Approach to its Treatment and Prevention."

Annual General Meeting of the Association.

The Annual General Meeting of the Association has been set down for Tuesday, 24th November, 1953, and the attention of members is drawn to this date. Full notice of the business of the meeting will be forwarded to all members.

EXECUTIVE REPORT.

Fact-finding Committee.

The tenth and final meeting of the Fact-finding Committee set up by the Minister for Health to investigate proposals made by dental technicians for direct contractual relationships with the public took place on Wednesday, 7th October, 1953.

In due course, all members of the Association will be supplied with a precis of the matters placed before the Committee by the representatives of this Association. This will be subsequent to the completion by the Chairman of his report on the deliberations of the Committee and its presentation to the Minister. All parties have been requested to avoid comment until such time as this has been done.

Country Convention—1954.

Following upon the marked success of the Country Convention held at Armidale in 1951, it has been decided that a further Country Convention will be held in Orange in mid-September, 1954. The tentative date is the week commencing 13th September.

The Western Division of the Association, having been granted Executive approval, are proceeding with the initial organisation of the Convention.

Members are asked to keep this date in mind. Preliminary publicity will be forwarded well in advance.

With the assistance of an appropriate committee, the Executive has established a set of rules for such Conventions and a series of suggestions for their organisation. It is considered that the holding of such Conventions in other than Congress years is of material benefit to the dental profession in this State.

Federal Office.

Matters of moment dealt with by the Federal Office of the Association are reported in a Newsletter published in this issue of the

Journal, so that no detailed report will be given here.

We would draw members' attention, in particular, to the information contained in this Newsletter concerning *Repatriation Dental Treatment, Pharmaceutical Benefits and Fluoridation of Drinking Waters.*

Post-Graduate Courses.

Acting upon a suggestion advanced by the Conjoint Meeting of Delegates from Divisions and the Executive, the Post-Graduate Courses Committee of the Association, at the Executive's request, is preparing a Post-Graduate Course in Pharmacology. The response from members to this course has been most gratifying.

The Executive is considering the possibility of a number of refresher courses from time to time in the basic medical and dental sciences and would appreciate from members an indication of their interest in such refresher courses, together with suggestions as to subjects.

Conjoint Meeting of Executive and Delegates from Divisions.

The Executive has given extensive consideration to various suggestions brought forward by this meeting. These included recommendations on such matters as Pharmaceutical Benefits, Country Convention, Dental Ethics and Survey Fund, Dangerous Drugs, Fluoridation of Drinking Waters, Divisional Finance amongst others.

The majority of the recommendations have been taken up by the Executive and now form part of its decisions and actions.

On investigation, several suggestions have been considered impracticable.

The suggestion that informative notices concerning candidates for election to the Executive be circulated to members was found to be empracticable under the Memorandum of Association of the Association.

The publication and sale to members of appointment books by the Association was found to be economically unsound.

Consideration was given to the amount of dental expenses allowed for taxation deduction purposes and it was felt that the present amount was equitable.

The Dental Journal of Australia.

The Executive has given consideration to future editorial policy in relation to *The Dental Journal of Australia.*

It has been decided that, as from 1st January, 1954, a salaried editor be appointed.

Attention is drawn to the advertisement appearing in the Classified Advertising Section of this issue of the Journal.

MEMBERSHIP.

New Members.

Akel, Frederick, B.D.S., F.D.S.R.C.S.; Bell, John Edgar, B.D.S.; Brown, Kevin Richard, B.D.S.; Clarke, Bruce Alexander, B.D.S.; Cutler, Robert Hamilton, B.D.S., D.D.S.; Dickinson, Allen Albert, B.D.S.; Dunn, Adrian Arthur, B.D.S.; Gadd, William Clark, B.D.S.; Hill, Walter Winser, B.D.S.; Humphries, Mervyn Osborne, B.D.Sc.; Nethery, Septimus James, B.D.S.; Rolfe, Cecil Granville, B.D.S.; Sanders, William Frank, B.D.S.; Shanahan, Noel Vincent, B.D.S.; Shanahan, Terence Patrick, B.D.S.; Tofler, Gerald Jachin, B.D.S.

Resumption of Membership.

Dr. J. K. Denney, Mr. L. S. Beckett.

Modified Leave of Absence.

Gillings, K. J. R.; Massie, J.

Leave of Absence.

Mallard, P. L.; Stern, B.

Deceased Members.

Baer, L.; Rinteln, J. E.; Yates, A. H.

Student Associates.

Brown, John Henry; Gray, Keith Thomas Crockford; Nader, Barry.

New Books and Publications

PARODONTAL DISEASE, by E. Wilfred Fish, London, 1952, ed. 2, Eyre & Spottiswoode. (252 pp., 37 illus.). Price 30s. (sterling). *Our copy by courtesy of the publishers.*

This revised and enlarged 2nd edition provides a complete handbook of Periodontia for the student and general practitioner, covering diagnosis, prevention and treatment.

The author has set down the results of his years of experience in diagnosis and tried and proven methods of treatment, in a practical easily understood manner. All aspects of Periodontia are discussed from diagnosis to pathology, and while one may disagree with the mechanics of treatment, no one can question the author's good faith.

A new chapter and appendix, with very good illustrations, have been added on "Occlusal Trauma and Parodontal Splints, and Partial Denture Design." There appears to be little difference in the basic design of splints and dentures: both are cast, tooth-borne, and incorporate occlusal support from all remaining teeth, thus making all teeth in each jaw work as one unit in mastication.

A timely opinion is expressed that more attention could be given in teaching schools to co-ordinating removable splints and partial denture design to periodontal treatment.

The importance of follow-up visits to ensure patient education and co-operation in post-operative care, is stressed throughout the work.

—*G. Morse Withycombe.*

COMPLETE DENTURE PROSTHESIS, by R. O. Schlosser and D. H. Gehl, Philadelphia, 1953, ed. 3, W. B. Saunders Co. (511 pp., 292 illus.). Price £4/0/9. *Our copy by courtesy of W. Ramsay (Surg.) Pty. Ltd.*

In this new edition the authors have endeavoured to bring up to date clinical and laboratory procedures. Greater emphasis has been placed on the importance of diagnosis and prognosis and considerable change is noticeable in secondary impression procedures and in the selection and arrangement of teeth.

The use of the newly developed dental materials is well covered in the subject matter of impression taking and tray preparation. The bibliography has been enlarged to include selected references to recent publications, in the supplementary reading for each chapter.

The authors have maintained the standard of the previous editions making this textbook most valuable for its unbiased coverage of the many aspects of complete denture prosthesis.

—*C. H. Graham.*

THE LAW AND ETHICS OF DENTAL PRACTICE, by R. W. Durand and D. Morgan, London, 1950, Hodder and Stoughton. (98 pp.). Price 7s. 6d. (sterling). *Our copy by courtesy of The English Universities Press.*

The authors point out in an "Authors' Note" that this book sets out to cover many common problems confronting dentists, upon which their advice has been sought in their capacities as Secretary of the Medical Protection Society and Deputy Dental Secretary of the British Dental Association respectively.

A number of chapters deal specifically with conditions of dental practice and the laws governing such practice in the United Kingdom and are, in consequence, of interest mainly to dentists practising in that country.

However, there are such chapters as those on Legal Obligations to the Patient, General Anaesthesia, Fractures and Dentures which have a general application wherever British law obtains.

The practical approach to dento-legal problems set out in these portions of the book is to be commended and will be of material assistance to the reader.

—*E. F. Hewlett.*

STUDIES IN THE MOBILITY OF THE HUMAN MANDIBLE, by Ulf Posselt. Supplement 10, Vol. 10, 1952, Acta Odontologica Scandinavica. *Our copy by courtesy of the editor.*

An investigation into the capacity of the mandible for movement in the occlusal and sagittal planes with special reference to retrusion, intercuspal and rest positions.

The methods employed comprise graphical registration and profile radiography, at vary ing degrees of vertical dimension.

The results tend to place some doubt on accepted definitions of rest position and centric relation in that it is shown that the movement from rest position to the intercuspal position is not a hinge movement. Further, it is demonstrated that the intercuspal position is not identical with the most retruded position of the mandible, being slightly anterior to it.

—*C. H. Graham.*

EXPERIMENTAL DENTAL CARIES IN ANIMALS: a historical review by Em. Stelling and Gosta Gustafson. Reprint from Odontologisk Tidskrift, Vol. 60, Nos. 1-2, 1952. *Our copy by courtesy of the authors.*

This comprehensive little publication is a review of the majority of the experiments relating to induced dental caries in animals. The authors have not attempted any explanation of the various findings that they have analysed, however this does not detract from the value of the monograph which should be of considerable assistance to those interested in dental caries research.

—*N. D. Martin.*

EXPERIMENTAL DENTAL CARIES IN GOLDEN HAMSTERS: by Gosta Gustafson, Em. Stelling and Edvard Brunius. Reprint from Odontologisk Tidskrift, Vol. 60, Nos. 1-2, 1952. *Our copy by courtesy of the authors.*

The problem of inducing dental caries in animals, both in relation to their diet and the type of lesion produced and the similarity to human dental caries has been partially solved by the use of the hamster, whose molar teeth cusps are covered with enamel in the same way as human teeth, and to whom a diet containing sucrose is highly cariogenic.

The authors have discussed an accurate method of estimating the score of carious lesins and have suggested that the best information on the increase in caries is given by sacrificing groups of hamsters after 70, 110, 150 and 190 days.

This report should be studied in conjunction with the previous publication on Dental Caries in Animals.

—*N. D. Martin.*

Information from the Dental Board

The following matters are reported from meetings of the Dental Board of New South Wales in May, June, July, August and September, 1953.

Applications for Registration

Akel, Frederick, B.D.S., University of New Zealand, 1945.

O'Riordan, Michael Henry, B.D.S., University of Sydney, 1951.

Restorations to Register

Cassidy, K. J.; Ellis, J. S.; Gadd, W. C.; Mathews, R. R. V.; Wherry, J. J.; Wooller, J. C.

Additional Qualifications and Descriptions

Callen, Kevin M., D.D.S. (Northwestern); Cassidy, Kenneth J., D.D.S. (Northwestern); Sullivan, Harold R., D.D.Sc. (University of Sydney) 1953.

An application for permission to use the description "Oral Surgeon" was deferred for further consideration.

Applications for Consideration

An applicant offering the qualification of the Dental School, Vienna, was referred to the Sub-Committee on Foreign Dentists for consideration and report.

Reciprocity of Registration

A foreign dentist was unable to satisfy the Board that complete reciprocity of registration existed between his country and New South Wales. In these circumstances, therefore, the applicant would be required to undertake an examination in terms of Regulation 28 in order to gain registration.

Advertising

A dentist who had advertised contrary to Regulations appeared before the Board to tender an apology and give an undertaking that he would in future observe the regulations in this regard.

Notification of Locum Tenens and Assistants

Advices were noted in reference to the employment of locum tenens and assistants.

Death of Mr. E. Cahalan

The President, Dr. J. S. Baird, reported the death of Mr. E. Cahalan, Q.C., a member of the Board, after a long illness. As a mark of respect the Board observed a period of silence.

Classified Advertisements

DENTAL RESEARCH OFFICER

The Medical Research Council of New Zealand invites applications for the position of DENTAL RESEARCH OFFICER. The successful applicant will work under the direction of the Dental Committee of the Council. He will be located in Wellington where he will be in a position to collaborate with various scientific and research organisations. Working accommodation is provided by the Dental Division of the Department of Health, with whom he will be closely associated. Certain field studies have already been made, and the Dental Committee has considerable data available related to the study of the etiology of dental caries. The primary task will be to continue these studies. The successful applicant will also be responsible for the dental aspect of an experimental fluoridation project sponsored by the Department of Health. He will be required from time to time to study and report on the clinical applications of laboratory research.

Applicants should be registered dental practitioners, and should preferably have a higher dental or other relevant qualification. Salary will be within the range £1,000-£1,500 per annum, the commencing salary being governed by age, qualifications, and experience. Reasonable travelling expenses will be paid. Applications, which close on 31st October, should be addressed to:

The Chairman, Dental Committee, Medical Research Council, P.O. Box 647, Dunedin, New Zealand.

PROFESSIONAL ROOMS

Rooms available. Kings Cross district. Excellent position. Suitable for second practice. Partly equipped. Ring FJ 3975, preferably between 8 and 11 a.m.

EDITOR, DENTAL JOURNAL OF AUSTRALIA

Applications are invited for the position of Editor to "The Dental Journal of Australia."

Appointment will be for two years at a salary of £250 per annum.

Duties will be to attend to the literary and editorial arrangements of the Journal, which is published each two months.

Further details are available upon request.

Applications should be addressed to the Secretary, Australian Dental Association, New South Wales Branch, and will close on Saturday, 28th November, 1953.

POSITIONS VACANT

LONDON. High class conservative practices, London and Reading (30 miles out), require two assistant Dental Surgeons, commencing November-December, 1953, for 1½ to 2 years. Good opportunity for keen, conscientious, recent graduates to gain experience and good remuneration. Write to Domb. and Bookless, 225 Shoreditch High Street, London. E.1.

TERRITORY OF NAURU
VACANCY FOR DENTAL OFFICER

Attention is drawn to the half page advertisement appearing on page xxvi of the advertising section of this Journal, calling for applications for appointment as Dental Officer in the Territory of Nauru.
Full details are contained in the advertisement.

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Use in Dentistry

OXYCEL designates oxidized cellulose prepared from various materials, such as gauze or cotton by a special process of oxidation which converts unoxidized cellulose into polyanhydroglucuronic acid—an absorbable haemostatic chemical.

OXYCEL, supplied as individual, sterile strips, effects prompt haemostasis when applied to oozing surfaces, and when left in contact with incised tissues is readily absorbed. When wet with blood it becomes slightly sticky and swells, forming a dark-brown, gelatinous mass. As soon as this discolouration occurs, bleeding usually ceases.

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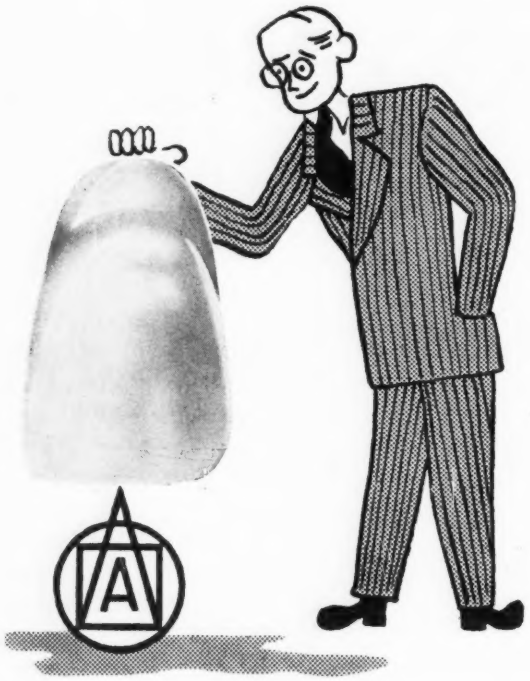

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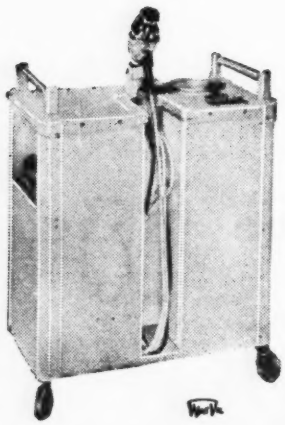

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The Kromayer Lamp is compact, convenient and has a definite place in Dental Surgery.

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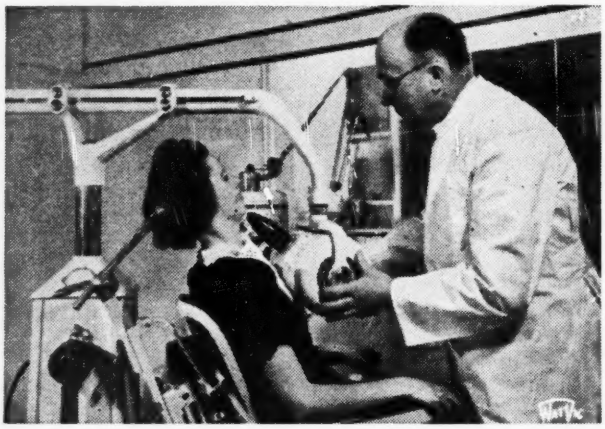

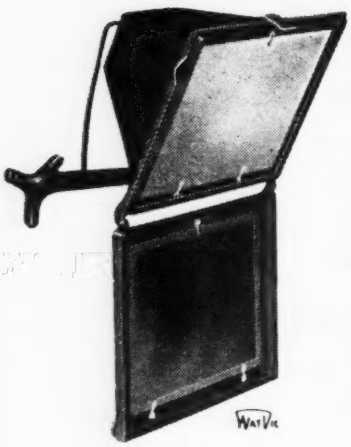

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● **Better Absorption**

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Added to enhance the therapeutic value of the tablet as the daily requirements of these factors are markedly increased during pregnancy and are often difficult to meet by dietary measures.

'Calvita'

EACH TABLET CONTAINS

Tri Calcium Phosphate	grs. 7½. (0.5 gm)
Ferrous Sulphate	grs. 1/6 (10 mg.)
Vitamin B1	333 int. units (1 mg.)
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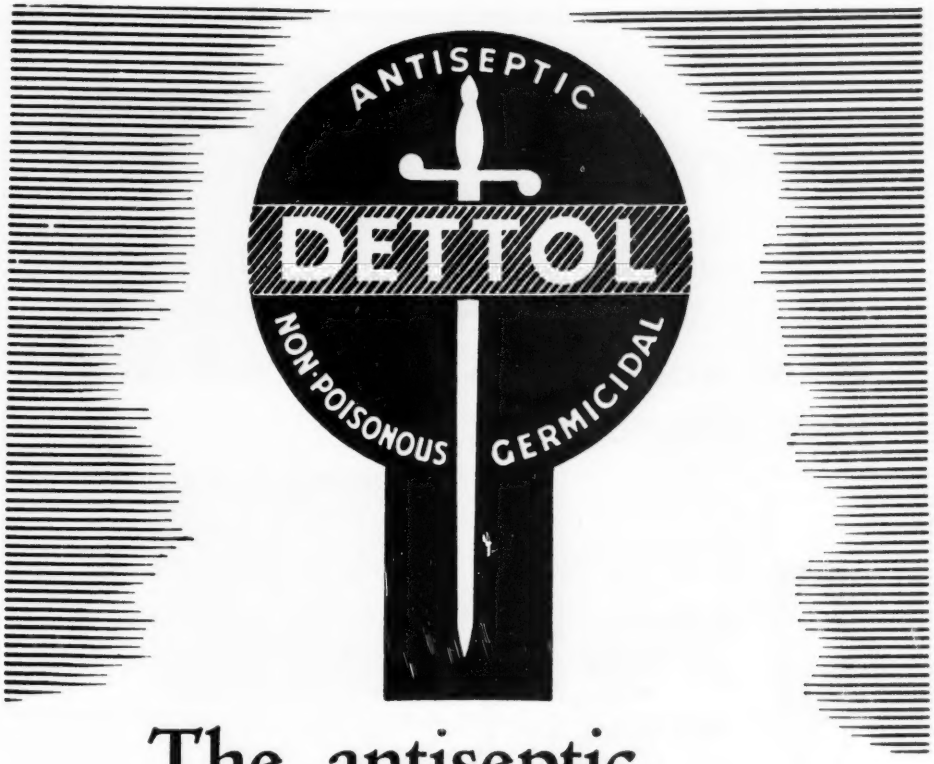

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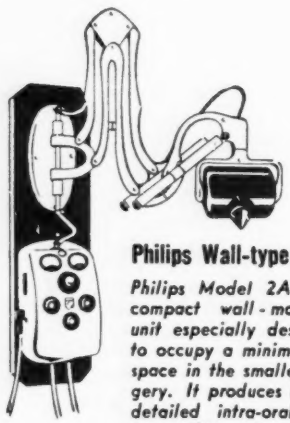
What would have been your diagnosis?



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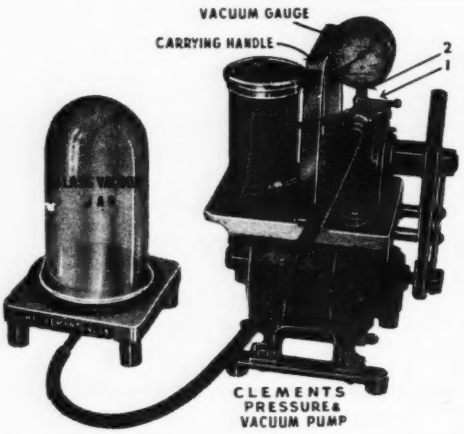

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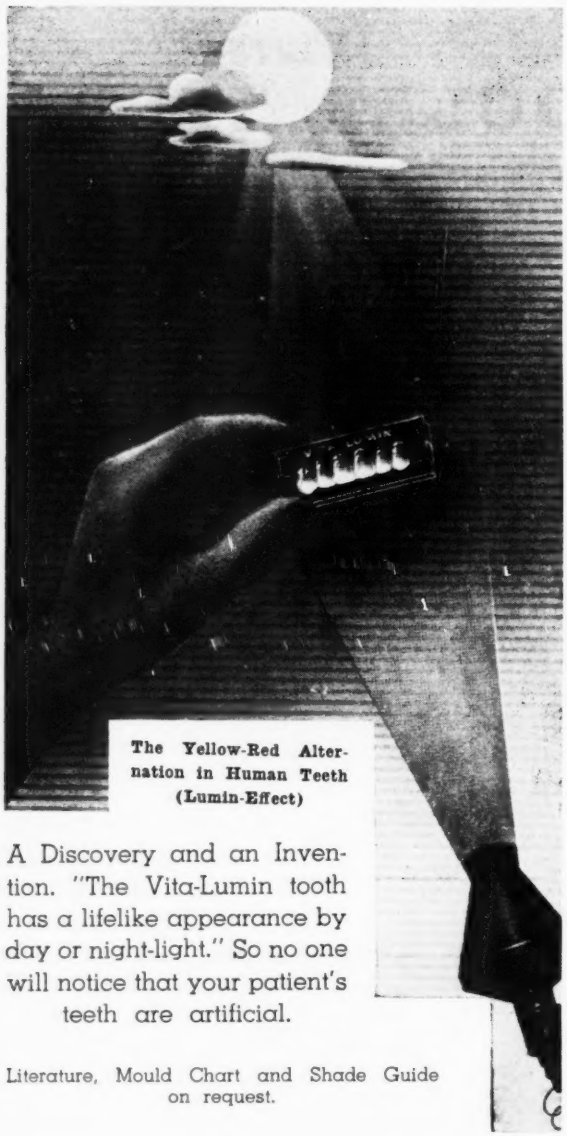

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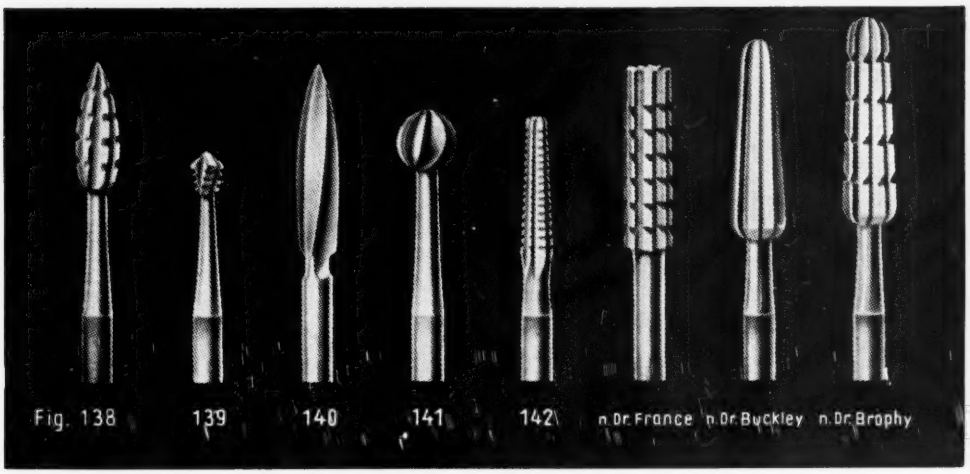

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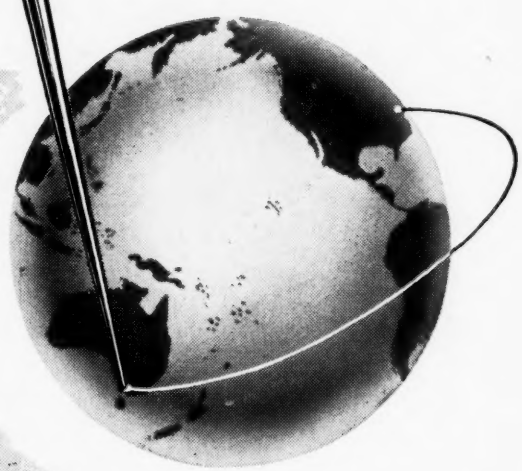

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